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The Hæmotoxic Nature of Eclampsia, with an Account of Fœtal and Placental Hæmolysins and an Experimental Investigation into the Anaphylactic Theory of Eclampsia.*

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To-DAY there is a growing tendency to regard the toxæmias of pregnancy as due less to one particular toxin than to a number. Eclampsia by itself has come almost to be considered as a group-name rather than as a separate entity.

Whitridge Williams,[1] for instance, strongly asserts that he does not understand how anyone, after examining the lesions found in fatal vomiting and in eclampsia, can attempt to assign to them a common origin.

Clinically, too, variation in incidence among other things has led many to subdivide eclampsia without, as I think, sufficient basis. For it must be particularly emphasized that the convulsion is not the essence of the condition. In Welch's[2] words, "one sees in print occasionally and hears of the expression 'eclampsia without convulsions,' but it is a very infrequent diagnosis because of the accustomed habit of regarding convulsions as the *sine qua non* of eclampsia." If one appreciates then that a woman may be fatally eclamptic (as judged on the post mortem room table) without convulsions, and that the convulsion is merely the very usual climax of an already present intoxication, post partum cases line up with the others. The important points are (1) the mental and general condition of the patient in the hours intervening between delivery and the onset of convulsions, rather than the length of that interval; and (2) how closely the percentage of cases in which a convulsion is the first symptom corresponds with such cases occurring before delivery. Unfortunately this last has not been elaborated as it might have been.

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Whitridge Williams, it is true, differentiates between cases without and with convulsions, and holds that the pathological lesions in the two are absolutely different. I hope, however, to show that the distinction between the two types of lesion which he describes is not so pronounced as he would make out, that it does not strictly hold good as regards convulsive toxæmia on the one hand, and non-convulsive on the other, and, further, that both may depend on the preponderance of one or other element in, or on the dosage as a whole of, the compound poison which is the subject of the first part of this paper. I do not say that the toxæmias of pregnancy are due to one toxin, and to one alone; but I wish to make it clear that *all* the lesions can be produced by one particular *type* of toxin acting under different circumstances. If this be so, then, until further data are forthcoming it is inadvisable to foster the view that eclampsia is merely a syndrome representing intoxication by a variety of poisons.

The type of toxin on which I am to enlarge is the hæmotoxin containing mainly hæmagglutinative and hæmolytic elements. On this point the first observation which I have found on record is by Flexner,[3] who, in 1902, drew attention to the fact that the minute thrombi so often found in the liver in cases of eclampsia are really hæmagglutinative in origin. They are due to a massing together of the blood corpuscles as the result of a circulating agglutinative toxin, and not to entanglement of corpuscles in a meshwork of fibrin. He found these capillary red-corpuscular thrombi in the neighbourhood of the areas of necrosis and hæmorrhage. Since then there have been scattered references in literature on the point, and it is now admitted by American, and less generally by German, pathologists, that the eclamptic necrosis is produced by an agglutinating toxin. Such a view, while correct so far as it goes, is, I take it, a narrow one if it does not lay stress on the essentially hæmolytic nature of the toxin.

In this country surprisingly little attention has been paid to such an idea. One still reads [4] of hepatic and renal eclampsias, of poisoning by autolytic products and of primary fibrinous thrombosis from increased coagulability. It will be of interest then to sum up the evidence in favour of such a view, the more so as it will, I think, be possible to add to that evidence in directions and on points hitherto neglected.

It may be well to detail the lesions produced by the inoculation of a definitely hæmotoxic poison, and then to compare them with those found in the toxæmias of pregnancy, and in particular eclampsia.

The very elaborate work of Pearce [5] on the appearances produced in dogs after such an inoculation is extremely interesting and instructive. In his experiments, the serum of rabbits injected with the blood or blood-forming organs of dogs was inoculated into dogs. If the animals did not succumb they were killed after 24—48 hours, which time was necessary for the production of a maximum of lesion.

The serum of a rabbit so immunized becomes moderately hæmolytic and highly agglutinative *in vitro* for the red cells of the dog.

The liver showed a marked necrosis—most commonly focal, but occasionally more diffuse—mainly in the periphery of the lobule. These necrotic areas were better developed towards the surface of the organ, and were definitely associated with hyaline thrombi in the smaller portal veins and in some of the capillaries. A deposit of fibrin was occasionally seen in certain of the larger thrombi, but the hyaline appearance was due to a simple massing together of the red cells without fibrin. Other capillaries were distended with formed corpuscles, while if the animal were killed before the onset of necrosis, intense congestion alone was visible. In some instances small hæmorrhages were found in relation to these necrosed areas. Such parts of the lobules as did not show necrosis exhibited a variable amount of granular and vacuolar degeneration.

That the necrosis was dependent on a real occlusion of the vessels Pearce demonstrated by ammoniacal carmine injections during life. The liver was the only organ showing these focal necrotic areas. This is ascribed to the slow portal circulation allowing the formation of larger thrombi. The better development of patches towards the surface of the organ (where the circulation is slowest) would confirm this.

The kidneys showed degeneration in the convoluted tubules, the spleen was enlarged and there was a hyperplasia of the abdominal hæmolymph glands. These little glands are peculiar structures containing the formed elements of blood and lymph intermingling in an adenoid reticulum with the formation of blood sinuses in place of lymph sinuses. Their function is not definitely known. They are numerous and well formed in the sheep and rat, and less well differentiated in the dog and cat.[6] All transitions are found from ordinary lymphatic glands to bodies closely resembling splenic tissue.[7] They are said to be numerous in man, and are best found in the retroperitoneal tissue. A careful examination of the glands from this region, which I made in 28 consecutive cases of post mortem examination, showed distinct transition stages of a rather poorly differentiated type in 18. The other 10 were ordinary lymphatic glands.

The work of Dudgeon, Panton and Ross[8] on the inoculation of hæmolytic and splenotoxic sera into rabbits confirms that of Pearce. They found necrosis of the liver, and also a degenerative change, but this latter was *quite independent* of the necrosis.

The kidneys showed fairly constant changes, especially in the convoluted tubules where the epithelium was swollen, degenerated or necrosed and the lumina obliterated or packed with red cells. A very interesting sequence is noted by them in the case of cats. They found that the kidney changes were extremely advanced, even in

animals dying in from 24—40 hours, whereas only when a fatal result was postponed for 56—76 hours did the liver show degeneration. The spleen was large, black and hæmorrhagic with the tissue cramped by the distended blood sinuses. Phagocytosis was extreme, the endothelial cells being packed with corpuscles or golden-brown pigment except in the Malpighian corpuscles. Agglutinated masses of corpuscles were met with occasionally, but in many cases the corpuscles were hæmolyzed. The mesenteric and groin-glands gave an appearance resembling a wasted spleen with open sinuses full of corpuscles and endothelial cells containing *débris* of golden pigment.

Such are the lesions produced in animals by the inoculation of a hæmagglutinative-hæmolytic toxin. It is easy to apportion to the two elements of this poison their respective share in the production of the lesions. With a moderate dose (whether quantitative or qualitative) no focal necrosis is produced, nor does it follow a serum with high hæmolytic and low agglutinative power. In the liver the peripheral necrosis following the thrombosis is certainly due to the agglutination as it occurs when dog's bile (which develops no hæmolytic properties in the rabbit) is the immunizing agent.[5] The degenerative changes, on the other hand, are due to the hæmolytic element, as they are readily produced without necrosis, or at most with necrosis of individual cells towards the centre of the lobule, by inoculating a purely hæmolytic poison, such as toluylendiamin, or by the daily injection in increasing doses of dog's serum into rabbits. The point to which I would specially direct attention, then, is that these two lesions are almost independent of each other.

The degeneration is usually better marked towards the centre of the lobule, and some diffuse necrosis may arise there if the toxæmia continue; but the degeneration does *not* proceed to peripheral necrosis. This results directly from a blocking of the contributory blood-vessels and not as the last stage of a degeneration from direct intoxication.

It will be observed, too, that these intoxications may lead, secondarily, to the formation of a real fibrinous thrombosis.

An examination of the organs in the toxæmias of pregnancy confirms the identity of the type of causative toxin. Viewing the matter broadly as a pathological picture, several points of comparison in the ordinary descriptions of these come forward which may be here considered with advantage—

(1) The liver lesions in both range from degenerative to necrotic changes.

(2) *The degenerative and massive necrotic changes are independent of each other.* In other words, the clinical severity bears no definite relation to the amount of necrosis discovered post mortem.

(3) The liver lesions in eclampsia resemble the experimental lesions produced by a poison whereof the agglutinative element is

sufficiently strong to resist the lytic element in certain peripheral vessels; but the liver changes in fatal vomiting conform more to a type where hæmolysis predominates.

(4) The limitation of the degeneration in the kidney to the convoluted tubules is marked in both. In neither, except in prolonged cases, is there the round-celled deposit of an inflammatory or reparative lesion.

(5) The kidney tends to be affected first.

(6) In both, cases occur wherein no fibrinous thrombosis can be demonstrated.

On this question of fibrinous thrombosis much discussion has been focussed. Schmorl,[9] whose work dates from 1902, insists on a primary thrombosis as the essential lesion, but most writers since then have been forced to admit a group where none is present; and others, as Edgar,[10] assert that when it occurs it is secondary. Undoubtedly fibrinous thrombosis *is* very frequent. But is not this to be expected when one recalls the factors inclining in that direction? These are (1) the hæmolysis going on in the small vessels; (2) the degeneration of the endothelium in the vessel walls; and (3) in eclampsia, the excess of carbonic acid in the blood. It has been suggested that the coagulation time of the blood is shorter in eclampsia than in normal pregnancy, and that this predisposes to fibrinous deposit. A lengthy series of experiments which I carried out some years ago completely confirmed Carstairs-Douglas[11] in his contention that this shortening does not occur either in eclampsia or the pre-eclamptic state. Only one of my cases showed a very definite shortening—and this in its terminal stages with deep cyanosis. The presence then of fibrinous thrombosis in certain cases of experimental hæmolysis and its occasional absence in cases of eclampsia would point to fibrin deposit being altogether secondary in nature.

A closer comparison of eclampsia with these experimental conditions will best be made by recounting shortly the appearances presented by three cases which I have had the opportunity of examining:—

Case 1. Primipara, æt. 18 years, full term, sudden onset, 14 fits, coma for 24 hours, duration 28 hours.

Abdomen: Small quantity of free fluid in peritoneal cavity; no visible hæmorrhages; veins distended with dark blood; large numbers of mobile glands in mesentery; duodenum and jejunum congested; ileum collapsed.

Liver: Normal in appearance but soft in consistence. Microscopically there is congestion in the portal areas; no inflammatory deposit is present in the connective tissue accompanying the portal vessels; a fibrinous thrombosis is seen in several of the larger ones but this is quite absent in *all* the smaller vessels; in these corpuscles are rarely visible but corpuscular debris is copious; there are no hæmorrhages. Nuclear staining is as a

whole well preserved throughout the organ; a little impairment is, however, noticable usually towards the centre of the lobule although in many of the superficial lobules the degeneration is better marked towards the periphery; the cells in these areas are separated and blown up and show vacuolar and slight granular changes. The point of most note is the extraordinary disparity between the *apparently* slight lesion and the clinical severity.

Spleen: Very congested and soft. Microscopically the spleen substance is rather wasted except around the Malpighian corpuscles, which are dense and prominent; congestion is pronounced and corpuscular debris is very marked in the vessels; an extensive thrombosis of the vessels is present. The endothelial cells are extremely active and contain much brown pigment; occasionally a red cell is definitely seen within an endothelial cell.

Kidneys: Normal in size; congested; capsules strip off readily. The microscope shows very marked congestion; a definite but not advanced degeneration is seen in the convoluted tubules and strictly limited to these; the epithelium is swollen and the nuclei stain poorly; the tubules are full of debris; there is no inflammatory deposit; the glomeruli and straight tubules stain well; no fibrin deposit is detected.

Lungs: Great extravasation into the alveoli and considerable thrombosis in the vessels.

Pancreas: Thrombosis present.

Heart: Some slight normal vacuolation present; no thrombosis.

Glands from the mesentery: simple hyperplasia of ordinary lymph-gland substance. (The retroperitoneal glands were not examined.)

Placenta: Nothing abnormal noted macroscopically.

Case 2. Primipara, æt. 21 years, 6½ months pregnancy, sudden onset, 13 fits in hospital (14 hours), comatose on admission, duration 30 hours.

Abdomen: A few drachms of free fluid; no congestion or distension of intestines; enlarged pink glands throughout the mesentery; several mottled glands (½–1½ cm.) in the retroperitoneal prevertebral tissue; three old calcareous glands were also present in the mesentery.

Liver: Rather small; superficial hæmorrhage, especially near the margins, none seen in the depths; congested as a whole. Microscopically there is congestion throughout the organ; numerous areas of necrosis are seen in association with the portal vessels and especially the smaller ones; these necrosed areas are with difficulty decolorised by Weigert's fibrin method and under moderate powers present an appearance somewhat resembling the stratified fibrin in the maternal spaces of placental tissue; higher magnification does not confirm this impression; they are larger and more frequent towards the surface of the liver where also some hæmorrhages are noted; apart from this necrosis, the lobules both superficially and deeply show a certain amount of degeneration—granular in character with occasional vacuolation and irregular nuclear staining. Formed corpuscles are found in relatively small vessels, but most of the small vessels are full of debris; occasionally a hyaline thrombus is seen in a peri-portal capillary or vein; a very few small vessels show a deposit of fibrin lining the lumen.

Spleen: Intensely congested—almost black; slightly enlarged; very homogeneous in cut section. Microscopically, congestion is extreme with some wasting of the spleen substance proper; golden brown pigment is present in excess either loose or within the large and active endothelial

cells; the Malpighian areas stand out and the nuclei show some disintegration; no fibrin is seen.

Kidneys: Normal in size; capsules strip easily. The microscope shows congestion and swelling, degeneration and feeble staining in the convoluted tubules; the glomeruli stain well; no thrombosis is visible.

Mesenteric Glands: Simple hyperplasia of lymph gland tissue.

Retroperitoneal Glands: These present a very peculiar appearance in marked contrast to the simple mesenteric glands; they are extremely congested and in places there appears to be a definite intermingling of the blood with the lymph stroma; the endothelial cells are extraordinarily numerous and many of them contain considerable brown pigment; the germinal areas are as a whole extremely prominent. While one cannot class these as well developed hæmo-lymph glands, still they strongly suggest an advanced transition form.

Placenta: Infarcts are unusually frequent; the syncytium shows some budding but scarcely out of proportion to the prematurity; sudan III staining is more positive than is usual with a 6½ months pregnancy.

CASE 3. Para, æt. 29 years, 7 months pregnancy, sudden onset, at least 10 fits, comatose on admission (30 hours before death), duration about two days.

Abdomen: Several drachms of free fluid in the peritoneal cavity; no congestion of intestines; mesenteric glands moderate in amount and size; several mottled retroperitoneal glands removed.

Liver: Not enlarged; some patches of superficial hæmorrhage. Microscopically there are areas of necrosis similar to case 2 but in most sections much more extensive; these areas, as in case 2, are associated with some hæmorrhage towards the surface of the organ, but the areas themselves are to be seen as widely developed in the deeper parts as near the surface; in addition a very distinct degeneration of the liver cells is seen extending usually throughout all the lobule and quite irrespective of the presence or absence of necrosis; in most it affects the lobule homogeneously but in a few it is better seen towards the intralobular vein; some sections show much less necrosis than others but the degeneration is equally well marked in all. The vessels are extremely congested and several hyaline thrombi are clearly visible; the capillaries are either packed with corpuscles or full of debris; the endothelium shows very little change even in the superficial areas with hæmorrhage, but there *does* here appear to be a little swelling and granulation. No fibrin thrombosis is seen.

Spleen and Kidneys: Deeply congested but not enlarged. Microscopically the appearances are as in case 2.

Retroperitoneal Glands: All are markedly congested; in the largest one (2 cm.) the structure is indistinguishable throughout most of its length from a typical and fully formed hæmolymph gland; blood corpuscles can be seen filling what in an ordinary gland are lymph spaces, and there is thus an intimate mixture of blood and lymphoid stroma; large areas closely resemble spleen substance; brown pigment is scattered throughout, especially in the endothelial cells. In one gland a very peculiar appearance was seen suggesting an aggregation of endothelial cells; these cells were certainly mesoblastic although at first sight the gland gave the impression of an invasion by carcinoma; high magnification showed fine strands of connective tissue running between them; these cells sheathed

in the lymphoid tissue on one side of the gland and sent short processes towards the centre where the tips had a certain resemblnace to the large cells of a Malpighian corpuscle; they showed no evidence of activity; I have not seen such a formation before and its nature I do not know.

The microscopical appearances in these three cases are strongly in favour of a toxin comparable to the immune sera of Pearce and Dudgeon, Panton and Ross. Case 1, where there was sufficient time for necrosis to develop but where none *did* develop, makes it apparent that the essential lesion is the degenerative one, and that focal necrosis, such as was seen in Cases 2 and 3, is more or less accidentally produced in that the hæmagglutinative element is powerful enough to produce an unyielding thrombus. With less agglutinin or more lysin these thrombi would either not have developed or would have been speedily broken up. The polymorphism of the lesion can then be mainly explained by the interaction of these two elements. Case 1 also, which resembles more the type of lesion found in toxæmic vomiting, confirms the observation that if the polymorphism of lesion in the toxæmias of pregnancy as a whole is due merely to dosage, or to variations in the proportion of these two elements one would not expect to find, and in fact does *not* find, any hard or fast lines of demarcation.

Peripheral necrosis of liver lobules, it must be remembered, is a far from common appearance. Opie,[12] in a study of 500 human livers, observed it in only four cases, and these were all associated with pregnancy.

There is, however, one additonal change usually seen in eclampsia to which reference must be made—multiple hæmorrhages. These are very prominent in some cases, especially in the brain and liver, and to an extent which would certainly indicate that the *convulsions* are not the cause. The brains were not examined in the cases I have described, but in the livers of Cases 2 and 3 only a few absolutely superficial patches of hæmorrhage were visible. They are to be explained by the presence of a third element in the poisoning—the endothelial toxin or hæmorrhagin. This toxin is not well developed in the lesion produced by Pearce, although small points of hæmorrhage in severe cases would make it probable that it is present. In other hæmolytic and agglutinative toxins (to which I shall refer under the heading of: The nature of the Toxin) it is much more readily detectable, and is apparently distinct from the toxin causing the degeneration of the liver cells.

The changes in the retroperitoneal glands seen in Cases 2 and 3 are interesting, and seem to add a link to the chain of evidence. It may be said that they simply represent hæmorrhages into the glands as the result of the endotheliolytic toxin. If so, then why do only these particular glands show it? With no knowledge of

them in normal pregnancy, and but little in other conditions, I must in the meantime let them speak for themselves.

The broad fact remains that all the types of fatal lesion seen in the toxæmias of pregnancy can be produced experimentally by one particular type of toxin, and that *a lesion apparently slight to microscopical examination merely indicates that a preponderance of the corpuscle-destroying element has led to the prevention of, or immediate splitting up of, all corpuscular thrombi and thereby prevented necrosis.*

Experimental work demonstrates also that a period of one to two days is necessary for the production of a maximum lesion after inflicting a *single* injury on the liver by a dose of serum. We have here surely an explanation of post partum cases of eclampsia.

I have gone into the histology of these conditions at considerable length because I think that an appreciation of these two processes at work in the liver will explain many of the anomalies of the chemistry of toxæmic pregnancy.

To put them briefly for the moment, the urinary changes in toxæmic vomiting are represented by an alteration of the ammonia-urea ratio with increase of the former; in eclampsia this alteration is not usually present.[14] How, then, is it possible to reconcile this difference with a common cause? By some the increased ammonia output is credited to the inanition resulting from the vomiting. It is not necessary, I think, to so ascribe the *whole* of the increase if a correct estimate of the vital changes in progress in toxæmic pregnancy is formed. These, as we have seen, are twofold and more or less independent—degenerative and necrotic. Although they are two stages in the process of cell destruction there is a fundamental chemical difference between them. A necrosed cell is one completely disorganized without power of repair and subject to immediate autolysis. No process of autolysis occurs in a denegerating cell. Vital processes are here in action, "the protoplasm is affected without the destruction of the nucleus. This change may set up enzymotic disturbances secondary in character, which are not connected directly with the actual life-processes of the cell, and which may readily return again to normal when the abnormal conditions of the cell are removed."[13] It thus becomes evident that deductions must be co-related to these two conditions, and, further, that investigations of the urine, however elaborate they may be, when unaccompanied in cases ultimately fatal by accurate histological reports, are of only moderate value, and are liable to misinterpretation. It is much to be regretted that in histological descriptions of these particular processes a certain looseness in terms should have crept in.

Again, if a certain proportion of cells have altogether escaped—if the lesion, in other words, be mainly focal—there may be sufficient

healthy tissue left to very considerably lessen the urinary evidences of the altered metabolism occurring in the affected parts.

With these preliminary remarks, I should now like to condense the results of Jackson and Pearce [13] in an examination of the urine of dogs in which had been induced those lesions so closely resembling eclampsia. The investigation was a thorough one, and of especial value in that the animals were brought to a state of nitrogenous equilibrium before inoculation.

In cases where post mortem examination showed diffuse degeneration without necrosis (*i.e.*, inoculation with a weak serum) they found the total N. and undetermined N. *almost* unaltered, or at most slightly increased, and a rearrangement of the ammonia-urea proportion whereby the former was increased and the latter diminished. This rearrangement may reasonably be put down to the altered functionating of *living* cells.

On the other hand, focal and diffuse necrosis presented a different picture. The total N. was increased, the ammonia percentage was slightly diminished at first, and then slightly increased, the production of urea remained *relatively* unchanged, while the undetermined N. was increased. This latter increase varied inversely with the amount of healthy tissue remaining, *i.e.*, the healthy cells could still split up the amino-acids and associated compounds.

Any comparison of these results with the urinary changes found by Whitridge Williams [1] and Ewing and Wolf [14] in toxæmic pregnancies must be on broad lines. For gaps inevitably occur: (1) none of the patients were, or were proved to be, in a state of nitrogenous equilibrium; (2) the post mortem reports are only two or three in number and very brief; (3) death or evacuation of the uterus frequently interrupted the purely scientific study; and (4) our knowledge of the nitrogenous metabolism of normal pregnancy is slight.

Ewing and Wolf preface their work with the following surprising statement:—"Although we have examined many apparently normal (*i.e.*, pregnant) cases, only two have been secured with approximately normal metabolism . . . and even in these the undetermined N. tends to run high, the ammonia also to a less extent and the urea low." Four other cases, clinically quite normal, are given where the same deviation from normal was present in a still more marked form, the total nitrogenous elimination tending to be very considerably less than in the non-pregnant state. Others [15] have shown a tendency in the same direction of nitrogenous retention.

After delivery a rapid return to normal takes place. There is also in their figures, and in those of Whitridge Williams, a decided rise in the total N. after abortion, so that figures for comparison with experimental conditions should be taken before that event.

Coming now to definitely pathological pregnancies, it will be

helpful to remember (1) that diffuse degeneration may be found in toxæmic livers without necrosis, but that necrosis, when present, is combined with a more or less diffuse degeneration; and (2) that the former is more representative of toxæmic vomiting, and the latter of eclampsia. In toxæmic vomiting the necrosis, when present, is not of the massive nature seen in eclampsia, but represents rather a necrosis of individual cells as the result of the acuteness of the degeneration. In cases, too, that recover, as the lesion is of the degenerative type, it is reasonable to suppose that necrosis may not have occurred to any great extent.

The main alterations in the urine in toxæmic vomiting as found by Ewing and Wolf are (1) a rearrangement of the ammonia-urea factor—the former being sometimes very high; (2) some increase of the undetermined N.; and (3) frequent decrease of the total N. *Even when this is normal, however, it may still be associated with a high ammonia coefficient.*

This, it will be seen, accords well with the results of Jackson and Pearce for the purely degenerative lesion. The comparison is not, of course, complete till one has brought into position the question of acidosis from prolonged vomiting. Until more figures are forthcoming for normal pregnancy it will be impossible to definitely settle how much of the excess of ammonia is due to this cause, but even if we put down part to it we are still left with a chart closely resembling the degenerative experimental lesion. Unfortunately no autopsy is recorded in the fatal case (No. 12), where the ammonia ratio was not increased at all, but it is very noticeable that as the case went progressively worse *the total N. and the urea increased.* This, as will be remembered, is more characteristic of a lesion where necrosis predominates.

The urinary changes in eclampsia and the pre-eclamptic state, according to Ewing and Wolf, consist of a reduction in the urea in proportion to the clinical severity, a variable ammonia coefficient—high only for a short time in cases that recover—and high undetermined N. In eclampsia the high undetermined N. did not seem to bear any relation to the clinical severity. Whitridge Williams notes a marked decrease in the total N., but Ewing and Wolf's cases do not altogether bear this out. In only two out of nine is the total N. less than the lower limit of " pregnancy values without symptoms," while several reach or pass the upper limit. It is also clear that the values for both the pre-eclamptic and the eclamptic are distinctly higher than those for toxæmic vomiting.

So far as a comparison is possible or justifiable, the variable ammonia, the high undetermined N., and the number of moderately high total N. values present a certain resemblance to the urinary findings in a lesion where necrosis predominates. The comparison is a more complicated one in this instance as the two processes of

degeneration and necrosis are more intimately associated. The lack of relation of the increased undetermined N. to clinical severity has the same significance as the lack of relation of necrotic lesion to clinical severity.

I do not wish to press these difficult comparisons unduly, but I think it will now be possible to give some pathological foundation for the empiric view of Whitridge Williams that, generally speaking, he regards a low ammonia value as a good sign in toxæmic vomiting and a bad sign in eclampsia. In the case of persistent vomiting a low ammonia value would indicate a slighter degree of upset of the ammonia-urea proportion—or, pathologically interpreted, less *degeneration*; whereas in eclampsia a low ammonia value would suggest that *necrosis* was very widespread, and a high one that a considerable proportion of liver cells was still alive and functionating albeit imperfectly.

Further, it will be seen that the gravity of toxæmic vomiting is not strictly to be measured by the excess of ammonia; for as necrosis advances the ammonia will tend to decrease.

On the question of *acid intoxication*, there can be little doubt, in view of the fact that Jackson and Pearce failed to find evidence of any of the supposedly poisonous acids, that it does not occur in the toxæmias of pregnancy, and that the *acidosis* so prominent in toxæmic vomiting is merely an associated phenomenon due to inanition.

Nature of the Toxin.

From the preceding it will be clear that I put forward the view that it is unwise as yet to dissociate in their causal relations pernicious vomiting and eclampsia. I have shown that the polymorphism of lesion and certain apparent anomalies in their chemistry, far from being an indication of diverse origin, can be readily explained by dosage or by interaction of the components of a toxin such as I have described.

The precise relationship of agglutinin to lysin is not yet clearly defined. It has been pointed out [16] that in immune sera they usually go hand in hand, although not absolutely so, as a high degree of lysis naturally tends to rapidly break up agglutinated corpuscles. Many consider that they are essentially distinct and separable, and this may prove to be the correct view.

Hæmagglutinins are not, I need scarcely say, confined to immune sera. Several of the higher vegetables (ricin, ergotin) and various bacterial toxins contain them.

If we now come to enquire more closely into the distribution and nature of such poisons, several points of interest appear.

Generally speaking, the work of Iscovesco [17] and others indicates that the whole group of hæmolysins and associated bodies is lipoidal

in character, or at least that hæmolytic action is represented by lipolytic.

An interesting comparison between the eclamptic toxin and various venoms may, I think, be drawn. The similarity of lesion is, indeed, most startling. All venoms contain the principles of hæmagglutination, hæmolysis and endotheliolysis. They have in addition a further element—the neurotoxic. The clinical course of toxæmic pregnancy certainly points to the presence of such an element. Venom toxins are lipoidal in nature; they act as true antigens—that is to say, they lead to the formation of antibodies—and their hæmolytic power is activated by lecithin.

In snake poisoning, the lesions vary according to which of the elements predominates. With Russell's viper, hæmagglutination is best marked; with the cobra, hæmolysis and neurotoxic effects. Wells [21] asserts that the efficacy of antivenin for neurotoxin and hæmolysin of *any* origin indicates that these substances are of a very similar nature, if not identical, in all snakes.

Nowak [22] has described in the liver degenerative changes with well-defined focal necrosis, and in the kidney acute parenchymatous alterations. Multiple hæmorrhages in the brain and elsewhere are of very frequent occurrence. Elliott [23] has noted a preliminary raising of the blood-pressure in cobra poisoning, and Noguchi [19] has studied the tendency to septic infection which follows. These, together with the extreme rapidity and completeness of the recovery which may occur, are features common both to venom poisoning and eclampsia.

As venoms are enzymal in origin (or closely allied thereto) this remarkable similarity would lead one to suspect that in toxæmic pregnancy a definite body of this nature is at work, and it is at least conceivable that research directed along the lines necessary for extracting these prolecithides would aid in the isolation of the type of poison responsible for these toxæmias.

Fœtal Hæmolysins

It has been suggested that some cases of eclampsia may be due to the fœtal serum acting as an immune or alien serum on the mother's system. The identity of the lesion in the organs of the mother and child by no means necessitates an intermediate source, as it is known that if the corpuscles of A. are hæmolyzed by the serum of B. the converse usually holds good.[24]

Dienst [25] claims to have found that in eclampsia the mother's serum *is* hæmotoxic to fœtal corpuscles. But, apart from this suggestion, the very marked differences existing between adult and fœtal blood make it of interest to settle the completely homologous nature of the two. The point of most marked similarity is found in the identity of their freezing points.[26] This indicates that osmosis

can occur equally readily in either direction. In other respects they differ markedly—in specific gravity, character of corpuscles, numbers of corpuscles, absolutely and relatively, coagulation time,[27] and natural hæmolytic power on animals.

Whilst one accepts the fact that iso-hæmolysins (*i.e.*, hæmolysins for homologous corpuscles) are not found in healthy serum, it is equally definite that they are developed in various morbid conditions.[28 29 30] On the other hand, iso-agglutinins are very frequently present in health in human serum, and anyone with much experience of opsonic work will have noticed the clumping of the red cells which sometimes occurs when serum is added. Hektoen [31] calculates that 90 per cent. of healthy individuals over three years of age possess iso-agglutinin for at least one of the three groups into which he finds it possible to divide all human bloods; while Landsteiner [32] has noted agglutination between the sera of mothers and the corpuscles of their children.

The experiments to be described were performed therefore to determine (1) the natural hæmolytic power of fœtal serum on animal corpuscles; (2) the presence or absence of iso-agglutinin in normal fœtal serum; and (3) the presence or absence of hæmolysin in the fœtal serum of diseased pregnancies.

Technique.

A length of umbilical cord was ligatured and cut off at delivery, wrapped in cotton wool and placed in an incubator till used. This was never later than 12 hours. Part of the contained blood was centrifugalized in Wright's tubes after clotting, and only tubes were used where clear serum was obtained. This is very necessary because, in cases where hæmolysis occurs, this process, according to Polk,[30] is greatly increased by solution in a serum of its own red cells. Washed red cells were prepared by dropping blood directly into citrated saline, washing three times in saline and standardizing as a five per cent suspension. Equal parts of serum and suspension were mixed in small dry sterile tubes with a diameter of 1 c.cm., incubated for two hours at 37°C., and then placed in an ice-chest.

Natural Hæmolytic Power for Animals.

This was investigated for rabbits and guinea-pigs. The hæmolytic power of normal adult serum on rabbits' washed red cells varies greatly. It is easy to demonstrate that different human sera act at varying rates on the same rabbit's corpuscles, and that the corpuscles of a series of rabbits are hæmolyzed at different rates by the same human serum. In various intoxications these differences are still more marked, diabetes, for instance, having a very high coefficient, and uræmia rather a low one.

The same variation in power was readily shown in the case of

fœtal serum. In general, however, the rate of hæmolysis was very much slower. Each of the 15 sera tested hæmolyzed 5 per cent. suspensions of the red cells of rabbits and guinea-pigs. The times taken to completely hæmolyze appeared to have no relation whatsoever to the times for the corresponding maternal sera. Halban and Landsteiner [33] have come to the same conclusion, namely, that the natural hæmolytic power of maternal serum is greatly in excess of that of the fœtus. Aschenheim [34] reports that fœtal serum does not hæmolyse sheep corpuscles till the eighth day, and is inconstant for a month, cow corpuscles after three weeks and swine only after three months. In the case of pigeons, the power is absent at birth in five cases out of six.

The opportunity was taken of comparing the hœmolytic power of 10 maternal sera on rabbits' washed corpuscles with 10 male and 10 non-pregnant female sera. No deviation from the normal limits was found.

The presence or absence of iso-agglutinins and iso-hæmolysins.

As the results were invariably negative both for agglutination and hæmolysis, they can be shortly summarized by enumerating the various combinations effected.

A. Experiments negativing the presence of fœtal iso-agglutinins for other fœtal bloods:—

(1) Normal pregnancies: 10 cases. Forty combinations of the serum of one fœtus with the corpuscles of another.

(2) Albuminuria: 1 case: severe with much œdema.
Fœtal serum—normal fœtal corpuscles (conversely also).

(3) Eclampsia: 1 case; moderately severe, 5 fits, recovery of the mother, death of 8 months' fœtus during labour.
Fœtal serum—normal fœtal corpuscles (conversely also).

B. Experiments negativing the presence of fœtal agglutinins or lysins for maternal or other homologous corpuscles:—

(1) Normal pregnancies.
Fœtal serum—maternal corpuscles: 9 cases (4 primiparæ, 2 with slight albuminuria).
,, ,, —male corpuscles: 4 cases.
,, ,, —non-pregnant female corpuscles: 4 cases.
,, ,, —Full-term non-maternal corpuscles: 4 cases.
,, ,, —corpuscles from 1 day post portum: 6 cases.
Maternal serum—fœtal corpuscles: 9 cases.
Full-term non-maternal serum—fœtal corpuscles: 5 cases.

(2) Still-birth: 2 cases; movements absent for 1 week and 3 weeks respectively before delivery at full term; no obvious evidences in the mothers or in the placentæ of the cause of fœtal death. In the absence of fœtal blood from these cases, normal fœtal

serum was combined with the maternal corpuscles and conversely also.

(3) Catarrhal jaundice: 1 case; death of the fœtus 1 day before spontaneous labour at 8 months.
Fœtal serum—maternal corpuscles (conversely also)
,, ,, —male corpuscles (conversely also).

(4) Albuminuria: 2 cases; severe with much œdema.
Fœtal serum—maternal corpuscles (conversely also).

(5) Eclampsia: 2 cases; both well-marked at 7½ and 8 months respectively.
Fœtal serum—maternal corpuscles (conversely also)
,, ,, —normal corpuscles 1 day post partum (conversely also).

In confirmation of the above results, guinea-pigs were immunized to fœtal blood by intraperitoneal injections of pooled blood from umbilical cords. The cords were bled directly into citrated saline—about 4–5 cc. (containing 2 parts of blood to 1 of diluent) being injected every 7–8 days. A pig was obtained whose serum completely hæmolyzed fœtal corpuscles (5 per cent.) in 20 minutes. Its hæmolytic power was tested against the following washed corpuscles (5 per cent.): male; full-term pregnancy; 2 days post partum: 2 cases; albuminuria 2 days post partum: 2 cases; catarrhal jaundice: 9 days post partum. Hæmolysis commenced in all from 5–12 minutes, and was complete in all in from 35–75 minutes. No agglutination was seen in any.

It would appear then that fœtal serum is wanting in iso-agglutinins, and that it does not develop iso-hæmolysins in diseased pregnancies. It cannot therefore in any way cause the hæmotoxic lesions found in pregnancy.

It will be convenient to mention here that in three cases of eclampsia I have failed to confirm Welch's [35] observation that the serum of eclamptics may hæmolyze normal red cells. Suspensions of pooled (5–6) blood were used, and in no case was any trace of hæmolysis visible.

The serum from Cases 2 and 3, described above, was contrasted with normal serum as regards power of inhibiting hæmolysis in a mixture of hæmolytic lipoid emulsion and red cell suspension. There was no loss of this power.

Placental Hæmolysins.

The results from the fœtus proving absolutely negative, an investigation of hæmolytic bodies from the placenta was next undertaken. Mohr and Freund [36] have shown that such can be isolated from normal placentæ. Etherial extracts of placentæ dried in vacuo yielded mainly oleate soap with, in addition, cholesterin and traces of a phosphorus-containing organic substance. The

hæmolytic action of the oleate was readily inactivated by maternal and somewhat less readily by fœtal serum. It is to be noted that the mere demonstration of hæmolytic substances amounts to little unless these can be shown to act as antigens. The following work has been a confirmation and extension of the work of Mohr and Freund, and has included comparisons of normal with eclamptic placentæ, as regards (1) the amount of ether-extractable lipoid present, and (2) the hæmolytic activity.

Nine placentæ were investigated—3 full term normal, 2 premature and 4 eclamptic. The lipoid was extracted from a convenient quantity of the whole placenta minced. Small pieces were preserved for microscopical examination—

		Total weight.	Lipoid from 20 grammes.
Normal:	No. 1	732 grammes	0·213 grammes.
	No. 2	510 ,,	0·171 ,,
	No. 3	737 ,,	0·170 ,,
Premature:	No. 4 (7½ mos.)	424 ,,	0·151 ,,
	No. 5 (6 mos.)	399 ,,	0·110 ,,
Eclampsia:	No. 6 (7 mos.)	481 ,,	0·179 ,,
	No. 7 (6½ mos.)	220 ,,	0·215 ,,
	No. 8 (full term)	450 ,,	0·318 ,,
	No. 9 (7 mos.)	170 ,,	0·264 ,,

Nos. 7 and 8 were noted to be rich in white infarcts; No. 9 contained several red infarcts. It will be seen that there is a definitely larger amount of ether-extractable lipoid in eclamptic placentæ.

The hæmolytic activity of the lipoids yielded some point of note. Varying quantities of a 1 per cent. emulsion were added to 3 per cent. suspensions of pooled red cells. The proportion required to produce complete hæmolysis in 0·5 cc. of suspension was as follows in each case:—

Full term normal:	No. 1, 0·5 cc.	No. 2, 0·8 cc.	No. 3, 0·5 cc.
Premature ,,	No. 4, 1·0 cc.	No. 5, 0·7 cc.	
Eclamptic ,,	No. 6, 0·2 cc.	No. 7, 0·06 cc.	No. 8, 0·1 cc.
	No. 9, 0·05 cc.		

Not only, it will be seen, did the eclamptic placentæ show more lipoid per given weight, but these lipoids were markedly more hæmolytic.

It now remained to determine if these lipoids were capable of acting as antigens.

A very pure lecithin, kindly given me by Dr. Hugh MacLean, of the Lister Institute, was employed to decide the question of

activation. A careful preliminary neutralization was necessary as the lecithin had become slightly acid, and thereby itself hæmolytic. No activation was induced by the addition of varying quantities of a 1 per cent. emulsion. One peculiarity was, however, noted. In several instances a very marked hæmagglutinative power was present. Addition of the lecithin absolutely prevented this. In the absence of activation I am not disposed to lay stress on this agglutination.

There is, then, no evidence of any prolecithide element in these lipoids—in other words, they were not found to differ from the ordinary lipoids of degenerating tissues. We are still left with the fact that in eclamptic placentæ the lipoids are present in larger quantity and are hæmolytically more active than in normal pregnancy.

It is possible that this increase is comparable to the increase of lipoid which occurs in the liver as the result of the hæmotoxic injections described at the beginning of this paper. The placenta is as rich in enzymes as the liver, and may suffer in a similar manner. On the other hand, Jackson and Pearce[37] have shown that as the fat content increases the iodine equivalent decreases. This scarcely tallies with the increased hæmolytic power of these eclamptic placenta lipoids, as the iodine equivalent is a measure of the lack of saturation, and therefore of the hæmolytic power. A decreased equivalent goes with *lessened* hæmolytic activity; yet in the eclamptic placentæ the reverse was found.

The Anaphylactic Theory of Eclampsia.

The peculiar condition known as anaphylaxis has received much attention in recent years. By it is understood the serious and frequently fatal attack, mainly convulsive, which follows the *second* inoculation under specific conditions of time and quantity of certain complex albuminous substances, not of themselves poisonous when administered in the same quantity as a solitary dose. The phenomenon has been largely studied by inoculating guinea-pigs with horse serum, but various other proteids, provided they are of *alien* origin, will produce it with great certainty—egg-white, milk and sterilized bacterial products. The less complex proteid substances (*e.g.*, peptone) are not so effective, while broken-down products, such as leucin and tyrosin, are ineffective.

The first dose takes 9–10 days to sensitize the animal. This is known as the refractory period, and the second dose, if administered within this time, proves non-toxic. At any later period it develops toxic symptoms. These usually occur very speedily, and may lead to death within five minutes, in which case convulsions are most pronounced. Under other conditions there is induced a train of symptoms, including cough, nasal irritability, ascending paralysis

and perhaps a few terminal convulsions; the animal may, however, recover.

Noteworthy points are: (1) that the proteid must be alien; (2) that there is a refractory period of 9–10 days, and that the serum of a sensitized animal will, on inoculation into another of the same species, passively sensitize it, *i.e.*, the animal is at once ready to receive the second or toxic inoculation.

Of the pathogeny we know but little. The process has some intimate relation to the development of immunity. The latest work [38] would show that the mechanism of "*immediate* death is an asphyxia from tetaniform contractions of the musculature of the smaller bronchioles. This is peripheral in origin, as it is developed in a pithed animal. Where death occurs more slowly it must be attributed to the pathological findings, which, shortly put, consist of marked degeneration endotheliolysis and multiple hæmorrhages, and so, *broadly speaking*, may be said to resemble eclampsia.

The first suggestion that eclampsia might be of this nature was given by Anderson and Rosenau.[39] Viewing the question from this standpoint, it occurred to them that the blood or some protein substances in solution from the fœtus or from the placenta might first sensitize the mother, and a subsequent introduction into the system of the mother of a similar substance explain the convulsions.

Their experiments with regard to the anaphylactic possibilities of fœtal blood were carried out on guinea-pigs. Pregnant and non-pregnant animals received a first, and, after an appropriate interval, a second injection of guinea-pig fœtal blood. Their results were invariably negative.

In the case of placenta, however, severe anaphylaxis was readily produced, but it is noticeable that the placenta, before inoculation, was allowed to autolyze for three hours at 37·0°C. This latter fact assumes much more importance when co-related to the work of Dryfuss,[40] who seems to have shown that autolysis takes place during life in the eclamptic placenta. A few experiments which I carried out seemed to conclusively prove that it was the autolytic rather than the purely placental element which produced the anaphylaxis. In the absence of material for the exact reproduction of their work, liver was substituted for placenta. Three guinea-pigs received intra-peritoneally ½ cc., 1 cc. and 2 cc. respectively of a *fresh* emulsion of one-half of a guinea-pig's liver, in 40 cc. saline and three weeks later 10 cc. of a *fresh* emulsion of guinea-pig's liver in 25 cc. saline, without obvious result.

On the other hand, two pigs receiving, at the same interval of time, the same amounts of liver which had been allowed to autolyze in an incubator for 24 hours at 37·0°C. showed undoubted anaphylaxis, consisting of cough, nasal irritability, respiratory embarrassment, paralysis of the hind legs, and, in one case, of slight clonic

convulsions. A control showed that the amount of autolyzed liver given was not directly toxic in a single dose.

Since Anderson and Rosenau published their paper several interesting communications have appeared, to only one of which for my present purpose it will be necessary to refer. In this the authors[41] seek to show that the serum of two eclamptic patients showed evidences of being sensitized to placental fluid or substance; in other words, could produce anaphylaxis *passively* in a guinea-pig. Serum from each case was inoculated intraperitoneally into guinea-pigs in quantity short of producing toxic symptoms. Two days later each animal received 6 cc. of normal amniotic fluid. Anaphylaxis was produced in each case. These experiments have, I consider, but little weight until it has been shown that serum from a normal pregnancy will not similarly sensitize. And, again, I have quite failed to produce any such condition. Two guinea-pigs were given intraperitoneally 3 cc. and 4 cc. respectively of the serum taken post mortem from the heart of a fatal case (No. 3) of eclampsia. Two days later each received 10 cc. of normal amniotic fluid, without result. A third guinea-pig, which received as a first dose 4 cc. of the fluid from the peritoneal cavity and the above dose of amniotic fluid, remained quite unaffected.

There is, then but little satisfactory evidence that eclampsia is anaphylactic in nature. Clinically, too, the picture of anaphylaxis does not greatly resemble it, and, convulsions apart, they have little in common. A guinea-pig with the paralytic train of symptoms most marked is alive to external stimuli, and will endeavour to crawl towards its cage while any motor power remains—a marked contrast to the dazed mental state of eclamptics. The pre-eclamptic state, also, has no counterpart in the anaphylactic condition; and, finally, undoubted anaphylaxis, as seen in the human subject, is very dissimilar to the laboratory picture.

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A Demonstration of Specimens Illustrating Cysts of the Female Appendages.*

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WITH NOTES.

By Alban Doran, F.R.C.S. (Eng.),
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Introductory Remarks.

Dr. A. Keith has kindly given me permission to publish this demonstration, and has revised the following report of it, taken from my own notes. The demonstration was founded on a series of preparations which I made between 1877 and 1884 for the Museum of the Royal College of Surgeons. Several have been described and figured in *Clinical and Pathological Observations on Tumours of the Ovary, Fallopian Tube and Broad Ligament*, and the drawings are here reproduced. The demonstration, however, specially deserves publication, not on account of the specimens, but rather for Dr. Keith's exposition of his theory of the homologies of the ovarian fimbria, which may clear up the mystery about the origin of the broad ligament cyst, a subject to which Coblenz, Bland-Sutton, Kossmann, Handley and myself devoted considerable attention in past years.—Alban Doran.

The Demonstration.

Professor Keith, after describing the preparations above alluded to, explained their nature by aid of a diagram (Fig. 1). The mesosalpinx (1) is bounded externally by a long process from the Fallopian tube known as the ovarian fimbria (8). If these parts be examined in an eight months' fœtus or in a new-born child, it will be found that ovarian tissue runs for some way up the ovarian fimbria, including that portion where the so-called "parovarian cyst" arises. This segment of the ovarian fimbria is part of the genital ridge. The Wolffian tubules (7) run towards the mesovarium at the point where the ovarian fimbria joins it, and they enter the hilum of the ovary. The Wolffian body represents the mesonephron, the kidney of the fish, and the mesosalpinx is the mesentery of the Wolffian body.

A process, often bearing a cyst, usually runs forwards from the

* Delivered at the College, April 22 1910.

Wolffian duct or "horizontal tube of the parovarium" (Fig. 2). What is it? Most likely it is a pronephros, as in *Myxine*, rather than a prolongation of the mesonephron or Wolffian body. Two or three pedunculated cysts, instead of one, are not rare at this point, and are known as Kobelt's tubes. A junction between the anterior part of the Wolffian duct and a process from the Fallopian tube is sometimes observed, as in this specimen prepared by Mr. Alban Doran, and figured in his *Tumours of the Ovary, Fallopian Tube and Broad Ligament* (Fig. 3). The process is of the type called by some writers a "Paratube." The significance of this junction will be discussed presently.

A pedunculated process at the ostium of the Fallopian tube, the "hydatid of Morgagni" (Fig. 1, 11) is simply an exaggerated fimbria (see Fig. 6).

Small cysts require close scrutiny as to their origin, for it is not certain that pathologists are correct about the starting point of broad ligament tumours. Dr. Keith examined 34 appendages bearing small broad ligament cysts, and in no less than 28 there was no evidence that they were of Wolffian origin. On the other hand, in the 6 remaining specimens, the small cyst could be seen arising from the Wolffian body. Mr. Doran's specimen here exhibited and figured in his treatise (Fig. 4), is undoubtedly Wolffian or "parovarian." It is, however, quite small, and Dr. Keith observed that cysts of this type could never be traced upwards, through series where the cyst grew larger and larger, to the common thin-walled broad ligament cyst, a kind of tumour very well known to the surgeon. With another type of minute cyst it is otherwise. In 20 per cent. of all subjects a minute cyst is found, connected with the ovarian fimbria. By comparing the male and female genital gland, Dr. Keith finds that the fimbrial cyst is developed from homologues of the rete testis. It is really in ovarian (hilum) tissue; the histology of the ovarian end of the ovarian fimbria has been already alluded to; that end is the representative of the rete testis. In the male, the tubuli seminiferi are continued as the rete testis in the border of the genital gland, and pass on through the old kidney, mesonephron or vasa efferentia to the vas deferens. In the female, there are tubules, which tend to become cystic, in the neighbourhood of the ovarian fimbria. They correspond in position to the rete testis. As in the male, these cystic tubules are quite distinct from the mesonephron, that is to say, from the "vertical tubes of the parovarium" (Fig. 1, 7). The vertical tubes pass on to the "horizontal tube of the parovarium" (Fig. 1, 5, 5), which is the beginning of the Wolffian duct, the homologue of the vas deferens.

Thus the parovarium or Wolffian body, though closely associated with the cysts (Fig. 5), so often detected at the ovarian termination of the ovarian fimbria, is not the parent of that type of cyst. On the

other hand, a series in the Museum of the College shows the gradual development of the minute fimbrial cyst into a large cystic broad ligament tumour, which, as has been explained, must *not* any longer be called parovarian. In all, however minute or bulky, the ovarian fimbria can be traced along the surface of the cyst, lying on the mesosalpinx. Compare this specimen (Fig. 6), where the Wolffian body or parovarium is seen to be quite internal to the cyst, with this example of a small but very typical broad ligament cystic tumour (Fig. 7). The course of the ovarian fimbria is plain, the alteration of relations to the Fallopian tube itself is caused by the opening up of the mesosalpinx by the growth of the cyst, which soon touches and afterwards stretches the tube itself. But even in the enormous broad ligament cyst (No. 4589 A), presented by Mr. Bland-Sutton, the relations of the ovarian fimbria to the cyst remain the same as in the most minute fimbrial cyst. The ostium of the tube opens on the surface of the cyst, in the characteristic manner long familiar to the pathologist, and from the ostium several low ridges of plicæ run along the cyst for over nine inches, ending on the outer pole of the ovary. Those ridges represent the ovarian fimbria.

We need not neglect the Wolffian body, epi-oöphoron, organ of Rosenmüller, or parovarium, simply because it is never the parent of a big cyst. It is large in mid-fœtal life and may have some function. The six small cysts above mentioned as undoubtedly Wolffian were all derived from the vertical tubes. Dr. Keith has never detected a cyst arising from the horizontal tube, more properly called the beginning of the Wolffian duct.

The paravaginal portion of the Wolffian duct, conspicuous in some of the lower mammalia, is the structure to which the name of Gartner, a Danish anatomist,[1] has been given. It is hardly correct to apply the name Gartner's duct to the free portion of the Wolffian duct between the Wolffian body and the uterus, sometimes persistent in our species (Fig. 1, 5′).

The inferior termination of the Wolffian ducts is not represented in *Homo* by Skene's tubes, but most likely by a pair of shallow depressions in the vestibule. Lewers operated on a cyst which, in his opinion, originated in the lower part of the Wolffian duct.[2]

1. Herman Treschow Gartner. He obtained a silver medal from the Royal Danish Society of Sciences in 1822 for a treatise, "Beskrivelse over et ved nogle Dyrarters Uterus undersögt glandulöst Organ." (Description of a glandular organ examined in the uterus of certain species of animals.).—A.D.

2. "Note on a Case of Cystic Tumour of the Right Broad Ligament springing from Uterus, and apparently developed from Gartner's Duct." *Proc. Royal Soc. Med., Obstetr. and Gyn. Section*, Jan. 1910, p. 67. (It was, if really of Wolffian origin, derived from the parauterine part of the Wolffian duct, not from the terminal or vaginal segment, "Gartner's duct." See also Amand Routh, "On Cases of Associated Parovarian and Vaginal Cysts, formed from a Distended Gartner's Duct," *Trans. Obst. Soc., Lond.*, Vol. xxxvi, p. 152).—A.D.

Vaginal cysts are not rare, but as a rule, for instance, in Mr. Doran's case, preserved in the Museum, they appear to arise from Skene's tubules which are the homologues of the prostatic tubules, since they are always developed near the urethra. We cannot, however, be too careful lest we should fall into error about homologies, because great proliferation of epithelium occurs in fœtal life, clogging the vagina. Hence the cyst above mentioned, and some other vaginal cysts preserved or reported, may have been developed out of epithelial invaginations.

Thus there is much doubt about the relations of vaginal and urethral cysts to the opening of the Wolffian duct. There is, on the other hand, no uncertainty whatever about cysts which develop higher up in the vagina; some of them undoubtedly arise from the paravaginal portion of the Wolffian duct, "Gartner's duct," [1] as it is generally called in human anatomy.

In order to perfect, if possible, our knowledge of the origin of these cystic structures, it is important to study the Woffian body and its duct in the human female independently of cysts and anomalies. The parovarium, as we usually call it, is not very small. Dr. Keith found that it can be most clearly defined in the new-born subject, although Percival Cole [2] recently reported that in the fœtus the parovarium can with difficulty be distinguished by the naked eye, and that at the age of twelve, although the uterus and broad ligament and all the sexual apparatus have considerably increased, the parovarium is still a structure which might easily escape notice. Keith, on the other hand, finds, as Cole did, that the Wolffian body or parovarium reaches its highest development quite late in adult sexual life, about the 35th year. Cole, indeed, noted the most evident examples in women from 40 to 43.

There are some fine dissections recently prepared for the College Museum demonstrating the Wolffian body in the human infant and adult, and its relations to the Fallopian tube and ovary. In one specimen a minute cyst is seen on the ovarian fimbria, and another on one of the vertical tubes of the Wolffian body or parovarium. As already shown, cysts of the latter type have never been traced upwards to the common broad ligament cystic tumour; there is little difficulty in demonstrating the development of the fimbrial cyst into the cystic tumour, a series of preparations at the College displaying that development.

The development of Müller's duct is not so intricate, but the

1. See Unterberger, "Durch Laparotomie gewonnene Gartnersche Cysten." *Monatsschr. f. Geb. u. Gyn.*, Vol. xxix, 1909, p. 587; an important paper, with references. An abstract appeared in the JOURNAL, Vol. xvi, p. 123 (August 1909).

2. "The Relation of the Parovarium to Cyst Formation." *British Med. Journ.*, Vol. i, 1910 (March 24), p. 748.

homologies of the terminal portion of the Fallopian tube are still disputed. Kossmann [1] and Handley [2] ascribe many cystic bodies and papillary growths in the region of the broad ligament to Müller's duct. Dr. Keith finds that about every third or fourth subject has a "paratube" or accessory tube, whilst he could only detect an accessory ostium in 1 in 40. Whilst Kossmann has so strong a belief in the Müllerian origin of many cysts, etc., recent embryologists have shown that the free end of the Fallopian tube is not originally a part of Müller's duct. Francis Balfour found that in sharks the fimbriated end of the Fallopian tube is a derivative of the head kidney or pronephros. This pronephros is a true excretory organ, it bears three or four tubules, and opens into the cœlom by a trumpet-shaped orifice, and is provided with a glomerulus which opens into the Wolffian duct. This may explain the double connection of the cyst already exhibited (Fig. 3), the junction of a "Kobelt's tube" with an accessory hydrosalpinx, for the terminal cystic process on the front of the horizontal tube of the parovarium (Fig. 2) is, as has been explained, most likely the relic of the pronephros and not Wolffian in origin.

Dr. Keith observes that accessory ostia are often blind, and tend to become cystic, as Kossmann and Handley have demonstrated.

Thus, after careful study of specimens and consideration of the anatomy and embryology of all structures concerned, broad ligament cysts may be classified as follows:—

1. The cyst of the lower end of the ovarian fimbria, originating in a homologue of the rete testis in the male, some tubular or cystic relic in the hilum tissue of the ovary which extends into this part of the ovarian fimbria. From this cyst arises the common cystic tumour of the broad ligament, the "parovarian cyst" of gynæcologists and surgeons.

2. The cyst of the Wolffian body, developed from one of its vertical tubes. This is, therefore, truly parovarian. No authentic instance of a cyst of this type becoming a large tumour has ever been reported.

3. The cyst of an accessory tubal ostium, the "accessory hydrosalpinx" of Handley.

Lastly, the "hydatid of Morgagni," and the similar pyriform pedunculated cyst running outwards from the horizontal tube of the parovarium are neither Müllerian nor Wolffian, but are relics of

1. "Anatomie und Pathologie des Nebeneierstöcke." Martin. *Die Krankheiten der Eierstöcke und Nebeneierstöcke*, Pt. iii.

2. "On the Origin from Accessory Fallopian Tubes of Cysts of the Broad Ligament situated above the Fallopian Tube." JOURNAL, Vol. iv (1903), p. 456.

the pronephros. To the same origin Kobelt's tubes may probably be traced. Dauber's case [1] may be of this type. Otherwise no instance of the origin of a large cystic tumour from an accessory hydrosalpinx or a "hydatid" of the fimbriæ or parovarium has been recorded.

1. "Case of Double Symmetrical Cystoma of Unusual Origin and Connections." *Trans. Obstet. Soc.*, Vol. xlvi, 1904, p. 341.

A DEMONSTRATION OF SPECIMENS ILLUSTRATING CYSTS OF THE FEMALE APPENDAGES.

By Arthur Keith, M.D., F.R.C.S. (Eng.).

ILLUSTRATIONS.

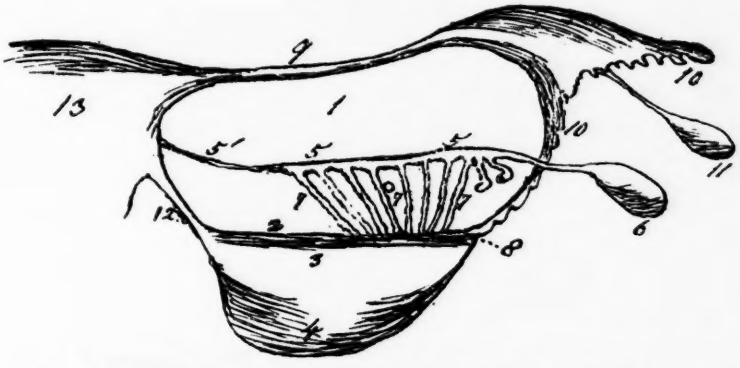

Fig. 1. Diagram of the Structures in and adjacent to the Broad Ligament.

1. Mesosalpinx.
2. Mesovarium (shaded line).
3. Hilum of ovary.
4. Ovary proper.
5. 5. Beginning of Wolffian duct, "horizontal tube of parovarium."
5′. Relics of free portion of Wolffian duct.
6. Cystic process from Wolffian duct, probably a relic of the pronephros, and not of Wolffian origin.
7. 7, 7. The Wolffian body, parovarium, epi-oöphoron, or organ of Rosenmüller.
8. Ovarian extremity of the ovarian fimbria of the Fallopian tube. It is partly made up of ovarian tissue, and includes tubular or cystic relics, homologues of the rete testis. The common broad ligament cystic tumour, the so-called "parovarian cyst," arises from a cystic relic of this type.
9. Fallopian tube, developed from Müller's duct.
10. 10. Fimbriated extremity of Fallopian tube surrounding cœlomic ostium, 11 "hydatid of Morgagni." N.B., 10, 11, and probably 6, are developed from the pronephric tubules and not from Müller's duct.
12. Ovarian ligament.
13. Uterus.

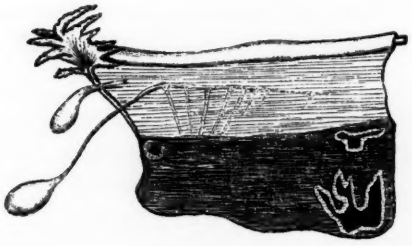

FIG. 2. A PEDUNCULATED CYST running forward from the Wolffian duct or "horizontal tube of the parovarium," probably not of Wolffian origin, but developed from the pronephros. The smaller cyst on a shorter pedicle may be of the same origin, or may be an "accessory hydrosalpinx" (Kossmann, Handley) of Müllerian origin. From Doran, "Tumours of the Ovary," etc., p. 46.

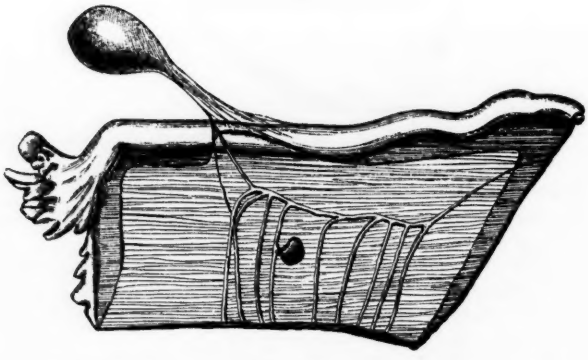

FIG. 3. WOLFFIAN BODY, PAROVARIUM, Epi-oöphoron, or Organ of Rosenmüller displayed by the removal of the posterior layer of the mesosalpinx. The free part of the Wolffian duct, running from the parovarium to the uterus, is unusually distinct. The vertical tubes are well-developed. The pedunculated cyst running forward from the "horizontal tube" is probably pronephric, as in Fig. 2; it is connected with the Fallopian tube, and thus may represent, according to A. Keith, the former close relations of the pronephros with the Wolffian and Müller's ducts. *Loc. cit.*, p. 43, Preserved Mus. R.C.S., Path. Series, No. 4586 A. (There is also a minute cyst connected with the Wolffian body and evidently of Wolffian origin.)

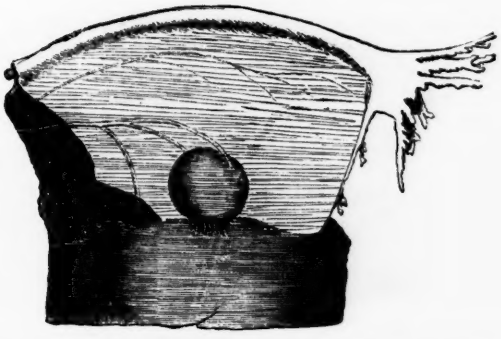

FIG. 4. CYST OF THE WOLFFIAN BODY or true "Parovarian cyst." The outermost vertical tube passes in front of it. The cyst is quite independent of the ovarian fimbria of the Fallopian tube. This true parovarian cyst is, according to A. Keith, not the origin of the so-called parovarian cystic tumour; it never attains a large size. *Loc. cit.*, p. 47.

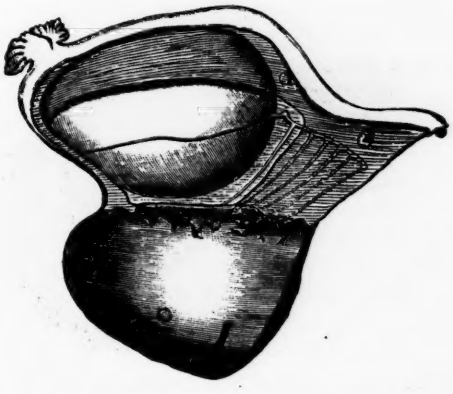

FIG. 5. WOLFFIAN BODY OR PAROVARIUM, with a thin-walled cyst developed in association with the ovarian fimbria, and not Wolffian. The outermost vertical tube of the parovarium crosses over the cyst and joins the ovarian fimbria, an unusual conditon; but originally the pronephros and Wolffian body are in close relation. The fimbrial cyst probably arises in a homologue of the rete testis (see text). *Loc. cit.*, p. 47.

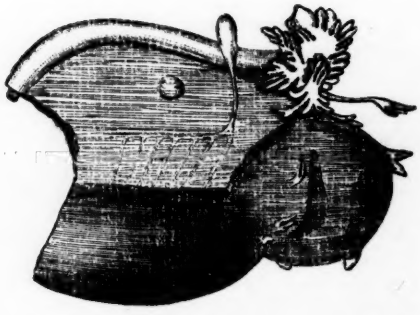

FIG. 6. CYST OF THE OVARIAN FIMBRIA. The secondary fimbriæ are stretched over the cyst and parted widely from each other. An incision made in the capsule exposes the cyst-wall. The capsule is formed from the mesosalpinx. A pedunculated pronephric cyst (see Figs. 2 and 3) springs from the Wolffian body. The "hydatid of Morgagni" (Fig. 1, 11) is here not cystic but fringed. The fimbrial cyst probably arises in a homologue of the rete testis. *Loc. cit.*, p. 53.

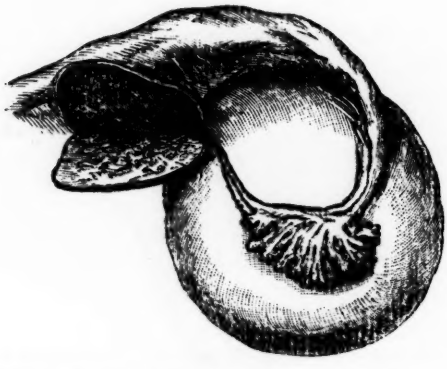

FIG. 7. A BROAD LIGAMENT CYST, incorrectly called "parovarian," but really arising in relics, homologues of the rete testis, at the point of junction of the ovarian fimbria and the ovary. It is in close relation to the ovarian fimbria, and through the opening up of the layers of the mesosalpinx it is beginning to come in contact with the Fallopian tube itself. *Loc. cit.*, p. 54.

On the Annual Variation of the Birth-rate in the Government Maternity Hospital, Madras.

By

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AND

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I.—STATISTICAL.

A CAREFUL and exact record has been kept of the number of confinements in the Hospital since its foundation in 1844. These have been tabulated in a form which shows the total number during any month of any year from September 1844 onwards. During the first 14 years of the Hospital's existence the total number did not reach 1,000 in any year. From 1861 onwards, however, the number of confinements has never been less than 1,000 a year, except in 1867, when it fell to 963.

Even a cursory examination of the data in this form shows that a very remarkable variation occurs in the number of cases from month to month which is repeated year after year. Take the year 1885 for instance; the number of cases is a minimum in February, and rises from that time to a maximum in October, when the number is about 45 per cent. greater than it was in February. From October the number falls off rapidly, and reaches a minimum again in February or March 1886; after that the usual rise is repeated. Changes of this kind take place regularly year after year, and the data show clearly that the number of cases undergoes a periodic variation, the length of the period being one year. The Hospital staff are, by experience, well aware of this seasonal variation; in September and October they expect the "rush"; in February and March the "slack time" comes.

Such a persistent and regular variation cannot possibly be a matter of chance; it must be the result of the action of definite physical agencies. It is therefore of interest to examine more closely the character of this annual variation in order to be able to decide if it be related or not to any climatic or other factors, and if these have influence on reproductive activity.

The first step in such an enquiry is to derive reliable mean values of the number of cases during each month. Fortunately the data available are sufficiently large, and extend over a sufficiently long period to enable us to obtain results which can be regarded with

some confidence as free from serious accidental irregularities which are superposed on and tend to obscure the variation in question. The following table shows the total number in each month for the five decades 1859–1908 inclusive, as well as the totals for each month for the whole period.

TABLE I.

	Jan.	Feb.	Mar.	Apl.	May.	June.
1859—1868	843	695	739	730	835	874
1869—1878	975	808	873	893	984	1,043
1879—1888	1,157	994	1,102	1,156	1,178	1,193
1889—1898	1,410	1,132	1,385	1,432	1,558	1,516
1899—1908	1,513	1,289	1,463	1,489	1,533	1,634
Sums (50 years).	5,904	4,918	5,562	5,700	6,088	6,260
Average for 1 year.	118·1	98·4	111·2	114·0	121·8	125·2

	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.
1859—1868	1,002	1,092	1,119	1,173	1,061	1,064	905
1869—1878	1,262	1,312	1,308	1,310	1,206	1,120	971
1879—1888	1,278	1,371	1,369	1,465	1,382	1,351	1,151
1889—1898	1,672	1,847	1,940	1,876	1,743	1,644	1,412
1899—1908	1,790	1,935	2,042	2,010	1,932	1,797	1,544
Sums (50 years).	7,004	7,557	7,778	7,834	7,324	6,976	5,983
Average for 1 year	140·1	151·1	155·6	156·7	146·5	139·5	119·7

The data for the first 14 years have not been included in the above, as for the greater part of this period the total number in each year was not sufficiently large to show with reliability the seasonal variation.

From the data given in Table I are deduced the average number for each month. The months are not all of the same length; hence the averages for the months would show differences from one another even though the rate were to remain constant throughout the year. In order to eliminate this effect, the daily rate is deduced by dividing the average number for each month by the number of days in the month. The values so obtained are strictly comparable amongst themselves, and are here taken to indicate the daily rate at the middle of each month—a series of approximately equidistant instants throughout the year. They are given in the following table:—

TABLE II.

Month.	Jan.	Feb.	Mar.	Apr.	May.	June.
Mean Daily Rate ...	3·81	3·48	3·59	3·80	3·93	4·17
Variation from mean for year = 4·32 ...	—0·51	—0·84	—0·73	—0·52	—0·39	—0·15

Month	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.
Mean Daily Rate ...	4·52	4·88	5·19	5·05	4·88	4·50	3·86
Variation from mean for year = 4·32 ...	0·20	0·56	0·87	0·73	0·56	0·18	...

The values given in the above statement do not represent the purely periodic or recurring change sought, but are affected by the progressive increase in the number of cases which is so clearly seen in Table I.

In Table I it will be noticed that there are fourteen columns, the second left-hand column being headed January and the fourteenth January also. The second column contains the total number of cases in the January months in the 10 years 1859–1868, etc., inclusive and the fourteenth column contains the total number in the January months of the 10 years 1860–1869, etc. Now had there been no progressive change, but only a purely periodic change, then the numbers in the second and fourteenth columns would have been the same. The difference between them gives the amount of the change, and knowing this it is possible to clear the values given in Table II of the effect produced by this change in the course of a single year—the assumption is made that the progressive change is uniform. The values of the daily rate for each month, and their variations from the mean for the whole year, thus corrected, are given in the following table:—

TABLE III.

	Jan.	Feb.	Mar.	April.	May.	June.
Daily rate	3·83	3·50	3·60	3·81	3·94	4·17
Variation from mean for year	—0·49	—0·82	—0·72	—0·51	—0·38	—0·15

	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Mean.
Daily rate	4·52	4·87	5·18	5·04	4·86	4·48	4·32
Variation from mean for year	0·20	0·55	0·86	0·72	0·54	0·16	...

The values given in the above table are plotted in Fig. 1, and a *smooth* freehand curve has been drawn to pass as closely as possible through them. The ordinate of this curve at any point denotes the variation of the rate from the mean at the corresponding time of the year.

Now this variation, shown graphically in Fig. 1 and numerically in Table III, can be shown to be very closely related to the annual temperature variation in the City of Madras, which has supplied the great majority of the cases recorded above. Two methods have been used to establish this relation. The first is a graphical method, the second is a numerical method; both lead to almost identical conclusions. The graphical method is the simpler and the more easy to follow, and will be discussed first.

The following statement gives the normal mean, maximum and minimum temperatures of the air at Madras for each month of the year, and the variations of each from its mean for the whole year. They are taken from the "Madras Observatory Meteorological Means":—

TABLE IV.

	Jan.	Feb.	Mar.	April.	May.	June.
Mean temperature	75·1	76·7	80·1	84·0	86·7	86·4
Variation	—6·0	—4·4	—1·1	2·9	5·6	5·3
Maximum temperature ...	81·6	86·6	89·2	92·9	97·8	98·3
Variation	—8·2	—4·2	—1·6	2·1	7·0	7·5
Minimum temperature ...	67·5	68·0	72·1	77·2	80·8	80·3
Variation	—7·2	—6·7	—2·6	2·5	6·1	5·6

	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Mean.
Mean temperature ...	84·5	83·3	83·0	80·6	77·5	75·5	81·1
Variation	3·4	2·2	1·9	—0·5	—3·6	—5·6	...
Maximum temp. ...	95·6	93·7	93·2	89·0	85·0	83·6	90·8
Variation	4·8	2·9	2·4	—1·8	—5·8	—7·2	...
Minimum temp. ...	78·5	77·3	77·1	75·2	72·3	69·8	74·7
Variation	3·8	2·6	2·4	0·5	—2·4	—4·9	...

The values of the mean temperature are plotted in Fig. 2, the time scale being identical with the time scale in Fig. 1, the scale of temperature being so adapted that the greatest variation from the average is represented by an ordinate of approximately the same length as the greatest ordinate of the curve in Fig. 1. A *smooth* free hand curve has been drawn to pass as closely as possible through these points. For the purpose of fixing the phase difference (by the method described below) the curve has been continued beyond

December into April again, the part from January to April being of course merely a repetition of the first part of the curve for the year.

On examining the curves in Figs. 1 and 2 we note the following similarities:—

Delivery Case-rate Curve.

1. The rate has one maximum value and one minimum value in the course of the year.

2. The rate *increases* from the minimum value in the first half of March to the maximum value in the first half of October, *i.e.*, during 7 months.

3. The rate *decreases* from the maximum in the first half of October to the minimum in the first half of March, *i.e.*, during an interval of about 5 months.

Mean Temperature Curve.

1. The temperature has one minimum value and one maximum value in the course of the year.

2. The temperature *decreases* from the maximum value in the beginning of June to the minimum value about the beginning of January, *i.e.*, during 7 months.

3. The temperature *increases* from the minimum in the beginning of January to its maximum in the beginning of June, *i.e.*, during 5 months.

These similarities suggest that the effect of (1) a high temperature is reflected at a later date in a low case-rate; (2) a low temperature in a high case-rate; (3) a falling temperature in an increasing case-rate; (4) a rising temperature in a decreasing case-rate.

These relations and the interval between any two corresponding states (say the maximum temperature and the following minimum case-rate) can be most easily and simply shown in the following way:—Take the sheet with the curve in Fig. 2 and apply it face downward, but without lateral inversion (*i.e.*, changing left to right) on to the sheet with Fig. 1. Adjust the sheet till the mean lines on the two sheets coincide (seen by transmitted light when held against the window). Now move the upper sheet to the left and it will be found that it can be so placed that the two curves nearly coincide throughout their courses. The close similarity between the two is very strikingly shown in this simple way. By putting the sheet with Fig. 2 face downwards on to the sheet with Fig. 1, we have formed what may be called an image of Fig. 2 in a plane mirror. This image is almost identical with the curve representing the variations in the case-rate at a later period.

When the curves have been brought into what is judged to be the best coincidence by the method just described, let a pin be stuck through the sheets at any point on the mean lines; it will mark two

associated dates on the two figures, and these two dates will enable one to determine after what interval (the phase difference) the case-rate curve repeats the inverted temperature curve. A trial will show that the interval is about 270 days. If the maxima and minima of the rate curve and temperature curve inverted be brought to coincidence, then the interval is about 260 days.

In Figs. 3 and 4 the maximum and minimum temperatures are plotted and a free hand curve drawn through them. The curves so obtained are similar in general character to the curve in Fig. 2. But when inverted and applied to Fig. 1 they do not show that close agreement with it that the mean temperature curve does.

The method just explained has the merit of simplicity, and is well adapted to visualize the relation between the temperature variation and the case-rate variaton. It is, however, not reliable enough to determine the phase difference with accuracy. In drawing the curves and in fitting them to coincidence something is left to the judgment, and habit may force a similarity where none may exist, or ignore it where it does exist. This will affect our estimate of what has been called the phase difference. It is therefore desirable to try and determine this difference by a more accurate, numerical method.

It has already been stated that the number of cases undergoes a periodic variation in the course of a year. If, however, the records for a single year be examined, this variation will not be shown correctly, because there is superposed on this natural variation other accidental variations which are irregular or non-periodic. Before we can get the true annual variation these irregular and non-periodic changes must be eliminated. The most serious of these have been eliminated by taking the mean values for fifty years. But to eliminate them all and completely by this method would require data extending over a very large number of years. The method of clearing the effect of the progressive change has already been referred to, and applied. In the graphical method the remaining irregularities were got rid of by drawing a *smooth* curve to pass as near as possible through the values when plotted (in Fig. 1) and taking this curve to show the course of the annual variation. The most probable form of the curve and the values of the variation at different times will now be determined by a numerical method.

Since the variation is periodic it can be represented by a complex harmonic function (Fourier's Theorem). Let V be the variation at the time t, then, according to the above,

$$V = p_1 \, Cos \, n\,t + q_1 \, Sin \, n\,t + p_2 \, Cos \, 2\,n\,t + q_2 \, Sin \, 2\,n\,t + \&c.$$
$$= U_1 \, Sin \, (n\,t + u_1) + U_2 \, Sin \, (2\,n\,t + u_2) + \&c.$$

where $n = \frac{2\pi}{\tau}$, τ denoting the period of the variation—1 year in this case. As the values of V are known at twelve equidistant instants throughout the year, the most probable values of the

constants (p_1 q_1) (p_2 q_2) (p_3 q_3), &c., or their equivalents (U_1 u_1) (U_2 u_2) (U_3 u_3), &c., in the above formulæ can be found by the method of least squares. The values of (p_1 q_1), &c., and (U_1 u_1), &c., so deduced are given in the following statement. The value of the constants of terms higher than (p_3 q_3) are too small to be taken into account:—

p_1	q_1	p_2	q_2	p_3	q_3
—0·323	—0·720	—0·130	—0·043	—0·005	—0·075
U_1	u_1	U_2	u_2	U_3	u_3
0·789	204°10′	0·137	251°42′	0·075	180°

The value of the variation in the rate at the middle of any month is given by either of these two equivalent formulæ—

$$V = -0{\cdot}323\ Cos\ 30°t - 0{\cdot}720\ Sin\ 30°t - 0{\cdot}130\ Cos\ 60°t - 0{\cdot}043\ Sin\ 60°t - 0{\cdot}005\ Cos\ 90°t - 0{\cdot}075\ Sin\ 90°t$$
$$= 0{\cdot}789\ Sin\ (30°t + 204°\ 10′) + 0{\cdot}137\ Sin\ (60°t + 251°\ 42′) + 0{\cdot}075\ Sin\ (90°i + 180°)$$

where t=0 for January, 1 for January, &c. The values of the variations for different months calculated by either of the above formulæ are given in the following table, in the row marked *C*. The original means (O) from which the constants of the formulæ were derived are also included in the table, while the row headed (O−C) gives the difference between the original means and the calculated values.

TABLE V.

	Jan.	Feb.	Mar.	Apl.	May.	June.
O	—0·49	—0·82	—0·72	—0·51	—0·38	—0·15
C	—0·45	—0·81	—0·76	—0·52	—0·36	—0·18
O—C	—0·04	—0·01	0·04	0·01	—0·02	0·03
	July.	**Aug.**	**Sept.**	**Oct.**	**Nov.**	**Dec.**
O	0·20	0·55	0·86	0·72	0·54	0·16
C	0·19	0·61	0·81	0·78	0·56	0·18
O—C	0·01	—0·06	0·05	—0·06	—0·02	—0·02

The differences are small, and are distributed almost evenly on either side. The values given in row C will now be taken as the most probable values of the variations of the case-rate at the middle of each month from the mean case-rate for the whole year.

In order to find the difference in phase between this series of changes and the mean temperature changes it is best to take some well-defined point on each, say the maximum or minimum. The dates of the maximum and minimum mean temperatures at Madras are given in the following statement taken from the "Madras Observatory Meteorological Means."

Highest Mean Temperature.	Lowest Mean Temperature.
26th and 27th May.	30th and 31st December.

The dates of the maximum and minimum values of case-rate can be computed from the following formula:—

Let u_a, u_b, u_c be three consecutive values of the variation near its time of maximum, the unit of time being the month. Then the time of maximum of minimum (x) estimated from the instant at which the variation is u_a is given by

$$x = \frac{-3u_a + 4u_b - u_c}{2(-u_a + 2u_b - u_c)}$$

Putting $u_a = -0{\cdot}45$, $u_b = -0{\cdot}81$, $u_c = -0{\cdot}56$ we get the date of minimum to be 1·42 or the 26th February.

Similarly the date of the maximum is found to be the 26th or 27th September.

The difference of phase can now be determined.

From the minimum of temperature to the following maximum of rate the interval is 271 days.

From the maximum of temperature to the following minimum of rate the interval is 276 days.

The difference of phase can also be determined by finding the times at which the case-rate variation is zero and determining the interval between these times and the corresponding times at which the temperature variation is zero. The times at which the case-rate variation is zero can be determined from the values given in table by the help of the difference formula—

$$V = u + \Delta_1 t + \Delta_2 \frac{t(t-1)}{2}$$

The times determined in this way are 1st July and 19th December. These give 263 days and 272 days as the phase difference.

Greater weight must be attached to the latter value, because the date 19th December has been determined from better or more favourable data, than the date 1st July. From June to July the change in the case-rate is 0·37, whereas from December to January it is 0·63. It follows that the effect of a small change or error in these differences will produce a greater change in the estimated

date at which the variation is zero, in the former case, than in the latter. Hence greater weight must be attached to the value 272 days.

Four values of this quantity have now been determined, viz., 271 days, 276 days, 272 days and 263 days. With the exception of the last quoted value they agree extremely well. The mean of the first three is 273 days. The mean of the four is 270 days.

The close character of the relation between the case-rate variation and the mean temperature variation can also be shown by another numerical method, which is free from subjective influences. For a description of this method, Vol. xx, Part 6 of the *Memoirs of the Indian Meteorological Department*, may be consulted, where a full explanation is given.

If we form, in the manner described in paragraph 8 of the above-quoted *Memoir*, a system of equations connecting the January case-rate variation, with the January mean temperature variation, etc., we deduce 0·122 (omitting sign) for the "correlation co-efficient," and ±0·192 for the probable error. If a system be formed connecting the January case-rate variation with the February mean temperature variation the correlation co-efficient is 0·354, and the probable error ±0·172. Proceeding in this way in search of the most perfect correlation and least probable error we get the following:—

Case-rate variation.	Temp. variation.	Cor.co-eff.	Prob. error.
January with	January, &c.	0·122	± 0·192
January „	February, &c.	0·354	± 0·172
January „	March, &c.	0·790	± 0·122
January „	April, &c.	0·980	± 0·008
January „	May, &c.	0·882	± 0·023

The correlation co-efficient is a maximum and the probable error least for the system formed from the January case-rate variation with temperature variation at some time in April, etc. The exact date in April can be determined from the values deduced above by the help of the formula already given on p. 262; the date so determined is the 20th April. Thus we have two dates—20th April for temperature variation, and 15th or 16th January for the case-rate variation giving a phase difference of 271 days. This is almost identical with the mean value of 273 days already found by the harmonic method.

Now if the case-rate in the Hospital be typical of the general birth-rate in Madras then the above analysis justifies the inferences—(i) that reproductive activity undergoes an annual variation; (ii) that this variation at any time is directly proportional in magnitude, but opposite in sign, to the mean temperature variation at that time of the year; (iii) that the interval after which the mean temperature variation is reflected in the birth-rate variation is about 273 days.

II.—Biological.

The vital statistics of the City of Madras show that the greatest number of births are recorded in the month of September; but the registration here is defective and inaccurate. The Madras Municipal bye-laws allow a period of six weeks between the birth of a child and its compulsory registration. The data cannot therefore compare in value with record of the Government Maternity Hospital. The statistics for the whole Presidency are so unreliable as to be of little use for the purposes of this investigation.

The Hospital record is a record of the deliveries of European, Eurasian and native women. The actual numbers for each of these classes cannot now be ascertained; but during the last five years the proportion has been approximately 80 per cent. natives and 20 per cent. Europeans and Eurasians. It has already been stated (p. 258) that the great majority of the cases in the Hospital are from residents in the City of Madras itself. The number of imported cases (*e.g.*, from other countries, and from Europe) are relatively too small to have any serious effect on the data.

The normal value of the period of gestation has not yet been satisfactorily ascertained. Physicians usually calculate that delivery may be expected in about 280 days after the last day of the last menstrual period. But it is now generally recognized that the time between insemination and actual fertilization of the ovum is an uncertain period varying from a few hours to 14 days or even more. There seems little doubt that whatever may have been the condition in primitive woman, ovulation and menstruation at the present time have practically no relation to one another in periodicity and date of occurrence. It has, however, been several times suggested by various authors that climate may have a direct influence on fertility. The discussion in Part I of this paper shows (p. 263) that the variation in the birth-rate at any time is proportional to the variation in the mean temperature at a time 273 days earlier. This can only mean that the variation in ovarian efficiency of the human female in proportional in magnitude, but opposite in sign to the variation of the mean temperature at the time, and that the period of gestation is 273 days.

From a physiological point of view the effect of a change in temperature is just as marked in natives as it is in Europeans. Anyone who has lived many years in Madras knows that natives dislike and feel the enervating effects of great heat almost to as great a degree as Europeans, and it appears that their reproductive efficiency is also as much affected by the seasonal change in temperature.

The fact that there is in Madras such a definite and well-defined

seasonal variation in reproductive activity would seem to help towards elucidating the question as to whether woman does not still tend to be monœstrous,* and whether there is still, in Madras at any rate, the remains of a distinct sexual season determined by climatic conditions. The community to which the data refer is of an extremely mixed nature, both in caste and blood; hence it is not possible to maintain that the seasonal variation is due to social customs or marriage seasons. Is it not possible, or perhaps even probable, that this annual variation is a vestigial remnant of a sexual season in the primitive human being? It seems reasonable to suppose that the curve can be more easily traced in those parts of the globe where extremes of temperature (with their attendant physiological effects) are experienced than in Europe and other temperate climates.

* W Heape, "Quarterly Journal of Microscopical Science," vol. xliv.

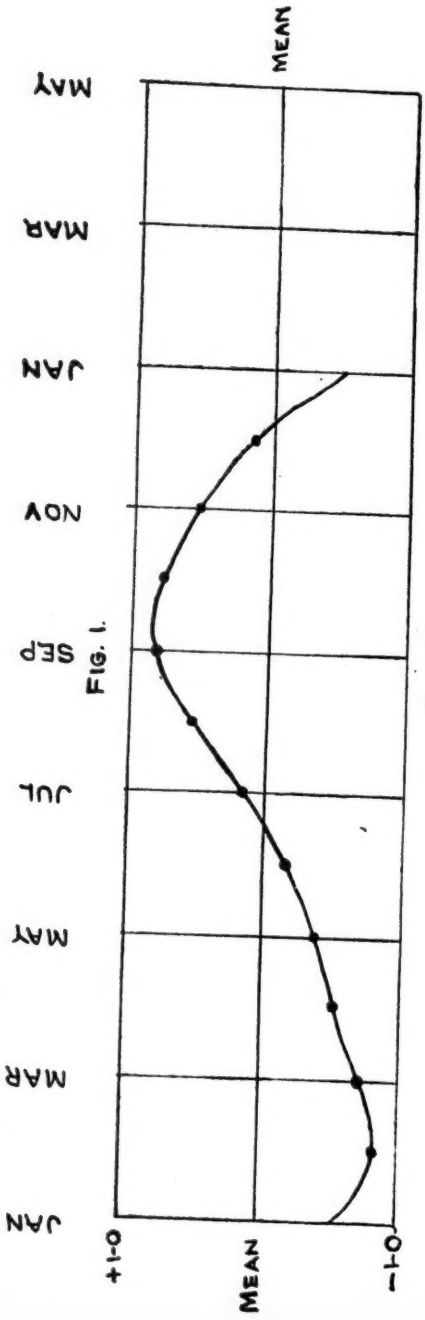
JAN
MAR
MAY
JUL
SEP
NOV
JAN
MAR
MAY
MEAN
+1·0
MEAN
−1·0

FIG. 1.

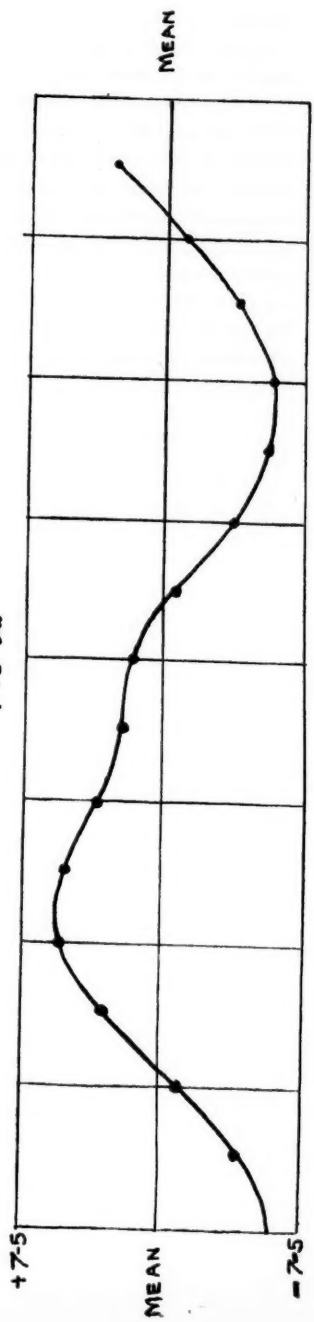
MEAN
+7·5
MEAN
−7·5

FIG 2.

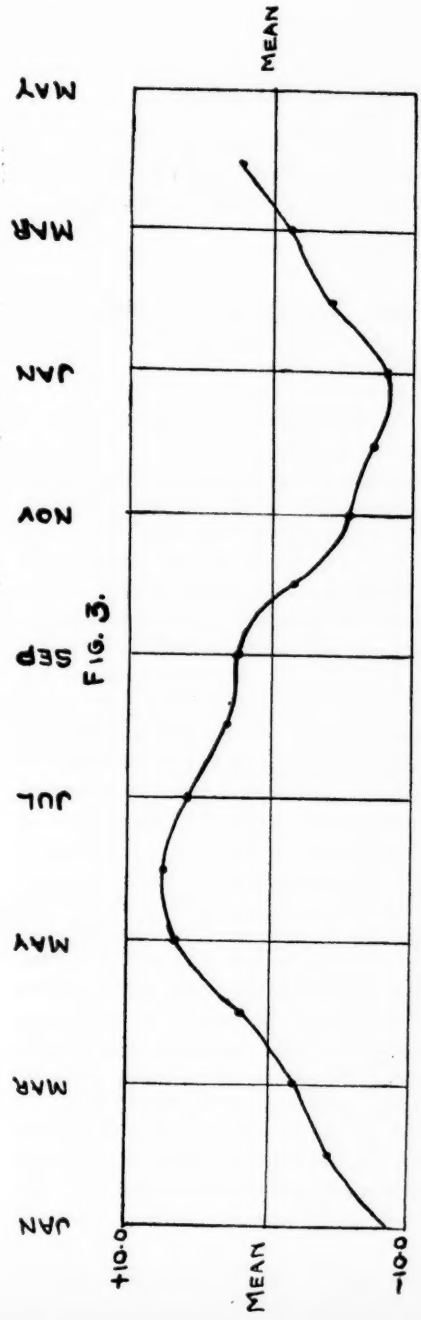
JAN
MAR
MAY
JUL
SEP
NOV
JAN
MAR
MAY
+10·0
MEAN
−10·0
MEAN

FIG. 3.

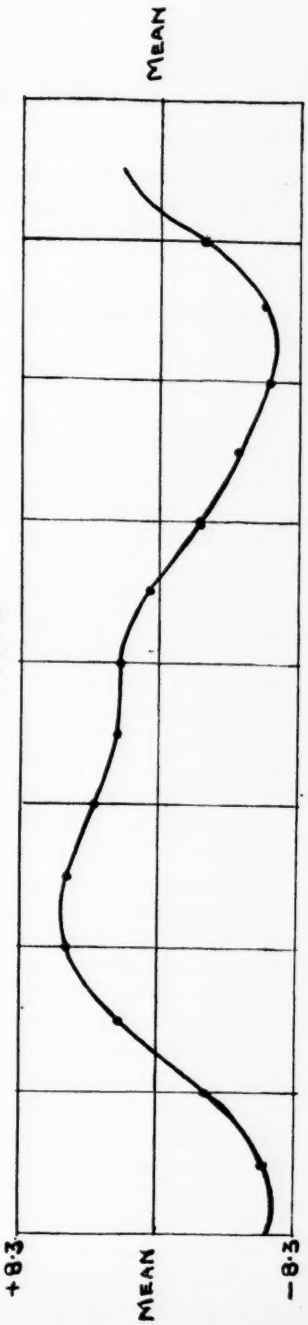
MEAN
+8·3
MEAN
−8·3

FIG. 4.

SELECT CLINICAL REPORT.

(Under this heading are recorded, singly or in groups, cases to which a special interest attaches either from their unusual character or from being, in a special sense, typical examples of their class).

Appendicitis Twenty-four Hours After Labour.

By Edwin Chill, M.D.

The following case of appendicitis after labour is worth recording as a rare complication of the puerperal state:—

Louisa B., aged 28 years, a thin, anæmic-looking woman, who had been married 10 months, was delivered of her first child on July 14 1909. The labour was quite normal till the head descended on the perineum, when, owing to inertia uteri, the patient was delivered by forceps, under chloroform. This was easily accomplished without perineal rupture. On the 15th, and as nearly as possible 24 hours after childbirth, the mother was seized with violent pains in the epigastric region, spasmodic in character, and accompanied by gaseous eructations. The temperature and pulse were normal, the uterus well contracted and free from pain or tenderness. A history was now obtained of similar paroxysms of pain at intervals for seven years previous to marriage, but that no such attacks had occurred during the period of pregnancy. The symptoms pointing to gastric or biliary colic, a hypodermic injection of morphia was administered, and the lower bowel was emptied of both flatus and fæces by means of a turpentine enema. In spite of this and the continuance of opiates with belladonna, the pains were but slightly relieved, and spread during the night from the epigastrium to the right iliac fossa. On the following morning the paroxysms were less severe, but the whole abdomen was tender (more so over the region of the appendix), distended and tympanitic; frequent vomiting and absolute constipation were present. Respiration was rapid, temperature 100°, and pulse 120. As the symptoms now pointed to acute appendicitis, followed by general peritonitis, she was removed to the Ealing Cottage Hospital, where the abdomen was opened, and the appendix, which was found thickened and perforated at its base, was removed by the honorary operating surgeon the day after the onset of the episgastric pains. The double event of instrumental labour and abdominal operation proved too much for the patient, and she died on the following morning.

TECHNICAL MEMORANDA.

(Under this heading will be published from time to time notes on points of practical interest in regard to methods of treatment, operative and therapeutic, and on the general management of Obstetrical and Gynæcological cases in hospital and private practice.)

Vaginal Subtotal Hysterectomy.*

By Arthur J. Wallace, M.D.,
Surgeon to the Hospital for Women, etc., Liverpool.

Whilst performing vaginal hysterectomy on account of non-malignant conditions of the uterus, the idea has several times occurred to me that removal of the entire uterus was a mistake, especially when the patient happened to be a married woman who was still under middle age. In such a woman the results of total hysterectomy are a weakened pelvic floor and a vagina of abnormal shape. It is needless to dwell on the first result. With regard to the second, it may be pointed out that the greatest capacity of the normal vagina is found in its upper portion. After removal of the uterus a gap is left in the pelvic floor and the resulting cicatricial changes compress the upper end of the vagina so that it becomes the least capacious portion of the canal. This alteration occurs independently of the atrophic changes in the vaginal walls due to the onset of the premature menopause.

In favour of the retention of the cervix there are further arguments related to the actual performance of the operation. A supravaginal abdominal hysterectomy is a simpler operation than total hysterectomy, a shorter time is needed for its performance and a smaller number of blood-vessels have to be controlled. Finally, fewer tissues are wounded and only removal of structures absolutely required is carried out. The same arguments can be adduced for vaginal subtotal hysterectomy, and here indeed they are even more cogent, because access is less easy and the operator works at a disadvantage. In abdominal supravaginal hysterectomy a few judiciously placed sutures effectively control bleeding because the field is immediately beneath the operator's eye, whereas in vaginal hysterectomy the bleeding points lie at the top of a tunnel.

* Read before the North of England Obstetrical and Gynæcological Society, May 6 1910.

In carrying out vaginal hysterectomy the writer has been in the habit of opening the utero-vesical pouch through the anterior vaginal wall and forthwith delivering the corpus uteri into the vagina. (The division of the posterior vaginal wall constitutes the final step in order to obviate the steady oozing of blood from its vessels during the operation.) The broad ligaments are then divided and sutured down to the level of the os internum where the uterine arteries bend outwards to cross the ureters. After ligature of the arteries (accomplished by direct, not mass, ligatures) the tissues remaining to be dealt with are the paracervical tissues and the posterior vaginal wall.

The paracervical tissue on each side of the cervix is a mass of firm connective-tissue on the anterior and posterior aspects of which run the vessels resulting from dichotomy of the branches of the cervico-vaginal artery (Byron Robinson). In the writer's experience, the controlling of these vessels is the most troublesome stage in the operation. Further, it is one of the most important, after the ligature of the uterine arteries, because post-operative bleeding appears to take place usually from either a cervico-vaginal artery or one of its branches. Such bleeding may have its source also from the posterior vaginal wall. Careful and accurate suturing of raw areas such as these consumes much time, and lengthens the duration of the operation. This expenditure of time and trouble would not be necessary if, when dealing with non-malignant conditions—apart from hysterectomy for prolapsus uteri—the corpus uteri alone were removed. Thereby trouble with vessels would be reduced to a minimum, and the operation would be shortened. Last summer (1909) this idea was carried out, and the operation proved to be a simple one.

The various steps have been performed as follows:—

1. The corpus uteri is delivered into the vagina.

2. The fundus uteri is pulled down and forwards, the vesical peritoneum is drawn forwards and sutured to the peritoneum covering the posterior surfaces of the supravaginal cervix and broad ligaments. The suture line runs obliquely upwards across the broad ligament to a point one-half to one inch external to the insertion of the Fallopian tube into the uterus. When completed this suture line closes the peritoneum, leaving ovaries and tubes intra-peritoneal and corpus uteri extra-peritoneal.

3. The broad ligaments are sutured and divided as far down as the os internum.

4. The uterine arteries are sutured and divided.

5. The corpus uteri is cut away from the cervix.

6. The cervical stump is grasped with forceps. Its anterior and posterior walls are apposed and sutured together, thus checking all possibility of bleeding from its vessels. Before these sutures are applied, if desired, the mucous membrane of the canal can be

excised, either separately or by cutting out a cone when dividing the corpus.

7. The broad ligament stumps are sutured to the cervical stump.

8. The vaginal wound is closed except for about 1 cm. of its extent which is left unsutured to allow of the insertion of a fine gauze strip into the potential cavity behind. The strip is removed at the end of 24 hours.

The operation has been carried out in but a few cases (chronic uterine bleeding, small fibroids, persistent and incapacitating dysmenorrhœa). In each instance it has proved to be easier and shorter than ordinary total hysterectomy.

REVIEWS OF CURRENT LITERATURE.

UNDER CHARGE OF EARDLEY HOLLAND, M.D.

LIST OF COLLABORATORS:

LONDON. Alban Doran, F.R.C.S.; J. J. Macan, M.D.; J. B. Banister, M.D.; J. Barris, M.B.; B. Glendining, M.D.; A. L. H. Smith, M.B.; F. E. Taylor, M.D.; C. White, M.D.; J. A. Willett, M.D.

EDINBURGH. E. Scott Carmichael, M.B.; Miss J. H. Filshill; R. W. Johnstone, M.D.

GLASGOW. E. H. L. Oliphant, M.D.

SHEFFIELD. Miles H. Phillips, M.B.

CHELTENHAM. C. J. N. Longridge, M.D.

The Influence of Prolonged Rest in Bed after Operations for Myoma, and after Labour, in respect to getting patients up sooner.

JOSEPH OPPMANN (I.D. Würzburg, 1910: *Muenchener m. Wchns.*, 1910, No. 31, S. 1661), on the basis of the experience of the Würzburg University Frauenklinik, declares that prolonged rest in bed is the best for the patients, and should always be ordained unless there is danger of hypostatic affections of the lungs. With this exception, early rising is unsuitable: (1) because it deprives the patients of rest that they desire and which they find does them good; (2) because it throws unnecessary work upon the organs on which convalescence depends; (3) because all its advantages may be obtained more easily and surely by comfortable measures not difficult to carry out; (4) because it has no effect in decreasing the morbidity, which can only be done by careful asepsis; (5) because it does not prevent thrombosis; indeed it cannot, as a rule, for other reasons be prescribed for the patients who get thrombosis. Early rising is, moreover, harmful (6) because it increases the danger of prolapse and hernia; (7) it favours infection, and, thereby, the danger of thrombosis; (8) because by its adoption in midwifery it may cause much mischief, and not only injure individuals, but strike a blow at the wellbeing of the population; and (9) because it rather provokes than prevents embolism. J. J. M.

Vaporisation (Atmokausis).

FLATAU, Nürnberg (*Volkmann's Samml.*, No. 585, 1910), sums up the pros and cons of this method of treatment. Dr. Flatau congratulates himself that he has little to withdraw from his judgments when he wrote the article in Sänger-Martin's *Monatsheft*. It is ten years since Pinkus published his method of applying steam, and Flatau gives his own experience. The disinfecting power of steam does not depend on its temperature or pressure, nor on the length of time of its application. The physical causes escape observation to some extent, but the more hyperæmic the mucosa the less is the action of the steam, and again the more fibrous the muscle the more destructive is the action of the steam. Dosage is accordingly difficult, and there is a long list of cases where this treatment of young women suffering from menorrhagia has resulted in absolute menopause with all its concomitant inconveniences. Disinfection and hæmostasis have fallen into disuse, but there remains another indication, the treatment of bleeding fibroids. Flatau limits the cases to those in

which the muscular wall is beset with numerous small tumours, partly interstitial, partly subserous, but with the uterus preserving so far its normal proportions. In no case must vaporization be employed if the tumour be markedly bulging into the cavity and thinning out the mucosa. These cases can usually be cured of the bleeding, but in no case does the myoma shrink, and in some cases the myomata may increase in size. It is accordingly advisable to try this treatment in patients who refuse operation, and it is of much use in patients who are too exsanguine to tolerate operative interference that can be postponed till recovery from the results of the hæmorrhage.

Sterilization, as advanced by Pinkus, is condemned by Flatau on the ground that so carried out it must be accompanied by a premature menopause with all its psychic disturbances; it is not to be compared to the easy and safe method of operating on the Fallopian tubes.

There remains the treatment of the hæmorrhages of the climacterium, and it is very important to remember that vaporization is to be employed only after an exact diagnosis has been reached by the examination of scrapings.

It is to be remembered that the steam does often set up inflammatory troubles, but these are caused by the lighting up of old, sometimes very old, septic foci; in such cases vaporization is contraindicated. The absorption fever which sometimes supervenes can be prevented on ordinary surgical principles—namely, by free drainage. This can be obtained by the insertion into the uterus of a glass drain, to be left in position for six days. This also prevents the formation of hydro- or hæmato-metra. Flatau was led to this by the examination of uteri in which he had experimentally applied vaporization at varying intervals before hysterectomy.

As regards technique, the simpler the apparatus the better, but as care must be taken that the inlet pipe be kept 3 cm. from the fundus, to avoid the risk of doing damage, Flatau fixes a collar on the tube. This will not pass the os externum, and can be so fixed as to keep the point at the required distance from the fundus. The vaporization should not last more than 20 or 30 seconds, and is best carried out from above downwards after the mucosa has been thoroughly dried. After curettage it may be necessary to pack with iodoform gauze. If the uterus be thoroughly dried, as it must be, the mucosa can always be destroyed. The usefulness of atmokausis is thus seen to be limited to a small number of cases of hæmorrhage. E. H. L. O.

Cold Vaginal Irrigation.

Alexandrow, Moscow (*Monats. f. Geb. u. Gyn.*, Bd. xxxi, Heft 4), considers cold as well as heat to be an important factor in the therapeutics of the female genitalia. Cold irrigations are appropriate whenever the action of the unstriped muscular tissue of the walls of the vessels or of the ligaments of the uterus requires stimulation. Hot injections which are a direct cause of arterial hyperæmia are suitable when we wish to increase the resorbent powers of the vascular and lymphatic system. To accelerate the regressive metamorphoses of childbed, cold irrigation is useful, as it is also in subinvolution of the uterus. J. J. M.

Dermatoses and Puberty.

Dalché and Fouquet (*La Gynécologie*, March, 1910) write on the cutaneous manifestations which are seen in girls at the time of the establishment of the menses from the 12th to the 16th years of life. They are somewhat similar, but more marked and more common than in boys at puberty. Some are physiological, such as the lineæ albicantes, the production of hair on the pubes and in the axillæ, the deposit of subcutaneous fat and the growth of the mammæ. So also with the development of sudoriparous and sebaceous glands.

Some of the pathological phenomena are mere exaggerations of physiological processes;

their etiology is doubtful, and internal secretion is often ascribed as the cause in addition to arthritis and other diatheses. Many of the dermatoses are most marked in girls suffering from amenorrhœa or dysmenorrhœa. A series of eruptions is taken up in detail with the appropriate treatment of each, such as hypertrichosis and the disorders of hair glands, seborrhœa, acne in its various forms, molluscum contagiosum, disorders of the sweat glands, urticaria and other œdematous conditions of nervous origin, adiposis, catamenial erysipelas of a recurrent type, pigmentary troubles, pruritus attributed to excessive internal secretions, herpes and so on through a long list of cutaneous affections. E. H. L. O.

Hypoplasia and Infantilismus in Obstetrics and Gynæcology.

A. MAYER, Tübingen (*Hegars' Beiträge*, Bd. xv, Heft 3), discusses the most important manifestations of infantilismus in the genitalia; anomalies of the perineum, hymen, urethra, vagina, portio, uterus in regard to anomalies of size, shape and position; also those of the ovaries, tubes, pouch of Douglas, urinary bladder and pelvis; and also of other parts of the body, of the head, ear, gums, jaw, teeth, neck, mammary glands, bony skeleton, skin and abnormal hairiness and intestines, and, finally, of the mental development. The causes of infantilismus and hypoplasia lie in arrested development, about the beginning of which we know nothing. We do not know whether the influence causing that arrest resides in the germs of diseased parents, whether they attack the ovum after impregnation and during embryonal or fœtal existence, or, finally, whether they only act after the child is born. The anomalies due to hypoplasia are discussed at the end of the article.

J. J. M.

Lacerations of the Deeper Parts of the Vagina.

NYSTRÖM (*Engström's Mitteilungen*, 1910, viii, 2,) omits tears resulting from coitus, already discussed by Lindström, as reported below, and those produced during pregnancy and labour. Lesions may occur from sudden stretching of the parts without direct violence, as in cases recorded where the women have fallen with the abdomen striking the corner of a step, or where the woman has fallen backwards twisting herself suddenly. The tearing may occur with a very moderate application of force, as in a patient who replaced her prolapsed uterus as she had often done before; on this occasion, however, it was more prolapsed than usual as the result of her efforts in carrying a heavy weight. The tear invaded the peritoneum resulting in protrusion of bowel, and the woman died. Similarly a tearing resulted from lifting heavy weights. More commonly the perforation has been caused by foreign bodies thrust into the vagina, either during a brutal assault, or from onanism or during manipulation to induce abortion. Of this sort was the case of a woman seen shortly before and immediately after the accident in the Helsingfors Clinique. The woman had borne six full term chifdren, the last 2½ months previously, which she was suckling, though she had menstruated. Examination showed a hyperinvolution of the genital organs. In spite of the diagnosis given the woman believed herself pregnant, and in trying to thrust the nozzle of a syringe into the uterus drove it forcibly through the posterior fornix up to but not through the peritoneum. The nozzle was of glass with smooth blunt edges, and about the thickness of the little finger. The wound was stitched and healed quickly. References to the literature are given. E. H. L. O.

Lacerations of the Deeper Parts of the Vagina sub Coitu.

LINDSTRÖM (*Mitteil. aus der Gynæk. Klinik des Prof. Engström*, 1910, viii, 2) brings together 80 cases, of which 3 are personal observations in the Helsingfors Clinique. The occurrence of such lesions has been denied by some medical jurists

and even by some gynæcologists, but there can be no doubt that they do result occasionally in apparently healthy women during normal coitus. The tear is in 50 per cent. of the cases found in the posterior fornix, and in only a few cases is it frankly in one or other lateral fornix. It may extend deeply into the cellular tissue, and may lay bare or even penetrate the peritoneum. The great majority of the lacerations occur in women of marriageable age, and in 35 per cent. of them the women had already borne children.

The etiology remains doubtful. Warman maintained that in all cases the cause was an excessive tension of the vaginal walls due to an extreme degree of sexual excitement on the part of the woman, but this cannot be universally true, for lacerations are found, though rarely, after rape; and in the second of Lindström's cases the woman had no sexual libido. The frankness of the woman's story made this statement readily credible. In many of the cases recorded the coitus took place in some abnormal position, with the woman half sitting or reclining, or in even more unusual attitudes. In the great majority of cases there is no disproportion of the genital organs nor any recognizable abnormality of the vagina. In a few there has been recorded some previous inflammatory process, in quite a number the intercourse was shortly after childbirth, and in one 3 months after a total hysterectomy. Cramp of the muscular wall may occur in vaginismus or in nervous women afraid of being caught in the act. There is apparently no single predisposing cause, but one or several of all these may be present. In all the cases recorded the hæmorrhage was excessive, but it can be easily controlled by plugging or in suitable cases by suture, and the prognosis is good, even when the peritoneum is torn; but of the recorded cases some have died of hæmorrhage and some of sepsis. A full literature is appended. E. H. L. O.

Hæmatoma Vulvovaginale.

J. Reich, Innsbruch (*Wiener kl. Wchns.*, 1910, No. 29), reports a personal observation of vulvo-vaginal hæmatoma, and discusses at length the mode of its occurrence during or after labour, either spontaneously or artificially, through excessive exertion or through the action of external force of any kind. In uncomplicated cases the treatment should be conservative expectancy, but interference may be indicated by great increase in the size of the swelling, which may even impede delivery, or by extreme pain, infection or fever. J. J. M.

An Enormous Labial Hernia.

O. Ulrich, Erfurt (*Zentralb. f. Gyn.*, 1910, No. 23), reports an inguinal hernia on the right side of a woman who had had 19 children; the length of the sac was 41 cm.; its circumferences measured at different points 31, 51 and 54 cm. She had never suffered from any symptoms of incarceration, but was affected by gastroptosis and gastrectasis of an extreme degree. J. J. M.

The Arterial Supply of the Lower Uterine Segment.

Couvelaire (*Ann. de Gyn. et d.Obstét.*, August 1910) has studied this question in four uteri removed soon after delivery. In two he injected the arteries with metallic suspensions, and then had them radiographed. The reproduction of the radiogram shows small branches going directly from the artery to the lower segment. Researches on the same subject have been made in eight cases by Davidsohn, Fredet and Schmorl; they also agree with Couvelaire—there would thus appear to be no confirmation of the statement made by Hofmeier that vessels to the lower segment go partly through the contractile part of the body of the uterus before reaching the inactive lower uterine segment. C. W.

Lutein Extract in the Treatment of Diseased Menstruation and the Premature Menopause.

ELLICE MACDONALD (*Journ. Amer. Med. Assoc.*, 1909, Vol. xlv, p. 205) has treated ten cases of premature menopause and ten cases of surgical menopause with a dessicated extract of the corpus luteum of the ovary of cows, obtained by skinning the outside of the ovary with a sharp knife in order to obtain as much of the essential substance as possible. In the ten cases of premature menopause, seven patients were benefited by the administration of extract of corpora lutea (grains 5) three times a day before meals. The earlier the case was treated the more definite seemed to be the results. The menstruation in the seven cases of improvement increased in amount and duration of flow. Two patients reported marked change in the character of the flow. In one very remarkable case the patient was brought back to the normal duration of flow, four days, while taking the lutein extract, and while untreated menstruated ten days. She was treated for three alternate months, and flowed four days, while at the three alternate months untreated she flowed two days. This seemed definite evidence of the effect of the corpora lutea in this case. Of the ten cases of surgical menopause, four reported some improvement, but it is doubtful whether this can be ascribed to the lutein extract. It seems that lutein extract is indicated in the cases of scanty menstruation and the premature menopause, but that the control of the surgical menopause by lutein is indicated after operation on pregnant women in whom miscarriage is feared. This is particularly true in the early weeks of pregnancy, during the embedding of the ovum, as it has been shown experimentally that the corpus luteum has a definite effect under such circumstances.

C. N. L.

Praeperitoneal Ventrofixation of the Uterus for Prolapse.

BOIJE (*Engström's Mitteilungen*, 1910, viii, 2) contributes a detailed statement of 112 patients on whom this operation was performed, and of 32 on whom a sero-serous suture was employed. All the cases are from Prof. Engström's Clinique in Helsingfors. The operation performed is somewhat similar to that introduced by Müller and practised by Kocher, and consists in laying bare the anterior surface of the uterus and stitching this with absorbable catgut to the bare recti; the fascia is stitched separately on the surface and then the skin. The results have exceeded expectations. Of 66 patients examined at periods of from nearly 6 years to 13 months after operation only one had relapsed, and that after three pregnancies. Pregnancy was not notably interfered with, and some of the patients had gone through many pregnancies. It is admitted that this operation will not prevent protrusion of the vaginal walls, and this must be attacked by a plastic operation at the same time as the ventrofixation. Hernia followed in a considerable number of the patients, some of whom had not been pregnant after the operation. The results of the sero-serous suture were not so satisfactory.

E. H. L. O.

Intraperitoneal Shortening of the Round Ligaments.

HIRSCH, Berlin (*Monats. f. Geb. u. Gyn.*, Bd. xxxi, Heft 4), whenever the abdomen is opened for other reasons, chooses for correcting displacement of the uterus, a proceeding which consists in dividing the round ligaments close to their insertion into the uterus, carrying the distal ends through the two folds of the mesosalpinx and stitching them to the posterior surface of the uterus.

J. J. M.

Laparotomy, a Retrospect of 1100 Cases.

NEBESKY, Innsbruck (*Volkmann's Samml.*, 1910, No. 587), gives an account of twenty years' experience in a provincial hospital with an epitome of his methods, indications for operation, and so on. He is conservative in his surgical operations, and is urgent on the necessity for laying all the immediate and ultimate risks of

operation before the patient that she or her friends may really appreciate the value of the procedure proposed. The paper is of interest as a record of the methods actually in use in a large provincial hospital at the change of the century. Special sections are devoted to the causes of death in general, after myomectomy and exploratory incisions. E. H. L. O.

Strangulation of the Duodenum after Abdominal Operations.

Engström, Helsingfors (*Engström's Mitteilungen*, 1910, viii, 2), relates two cases of this dangerous and ill-explained complication from his own experience. In the first case the diagnosis was unfortunately verified by autopsy; the patient was a woman of 48, nullipara, who was submitted to myomectomy by subtotal hysterectomy. The operation was easy, and the recovery was apparently established, but from the first day the case book records that the lower abdomen was indrawn. Flatus and fæces were passed early, but on the fourth evening after the operation the epigastrium began to be distended, and there was a little regurgitation of sour water from the stomach. This was succeeded by excessive distension in the epigastric region with vomiting of food and yellow fluid, which last was in excess of the fluid swallowed. The area of distension gradually extended downwards, but was always sharply defined from the retracted area. On the 6th day the conditions were aggravated, and the stomach was washed out, but the patient collapsed and died. The necropsy showed the organs generally to be healthy, with exception of the heart, which was fatty. The stomach was enlarged moderately, and the walls not markedly thinned but congested. The pylorus was open, the upper part of the duodenum was free with a long mesentery, and its diameter was enlarged, till it passed under the radix mesenterii, where it was sharply kinked and occluded. From this spot downwards the whole bowel was collapsed. Before the operation the patient had suffered from digestive troubles and at first thought little of her symptoms after operation. The other case was that of a woman, of 69, suffering from an ovarian adenocystoma. She was emaciated and feeble, but her organs were healthy. The tumour was easily removed. On the 4th night after the operation the patient began to vomit, and the epigastrium was much distended. After a short remission the vomiting recurred, the ejecta being at first green, then coffee-ground, but never fæcal. A stomach tube removed about a litre of coffee-ground fluid, and this immediately relieved the woman, who was placed for 10 minutes in the knee elbow position. She made a complete and rapid recovery. In both cases an increase in the pulse-rate was the first note of warning; the temperature usually remains normal, though there may be a little fever in the last stages. The strength ebbs rapidly, and the patient becomes apathetic. These two cases are typical, and it may be that this accident is more common than is generally believed as they are sometimes diagnosed as peritonitis. Engström reviews the literature of the subject. The tightness of the radix mesenterii is considered to be the cause of the condition, and in his cases there was certainly no atony of the stomach. The knee elbow position allows the collapsed bowel to rise out of the pelvis and relieve the tension on the mesentery. Experiments on the cadaver have shown that traction downwards of the small intestine kink the bowel at the junction of the duodenum and jejunum by drawing it against the spinal column. This condition readily occurs when the emptied bowel slides into the pelvis to occupy a space previously occupied by a tumour, and Engström suggests the knee elbow position assisted by a speculum in the vagina to admit air. The loss of fluid into the stomach must be compensated by injections into the bowel or subcutaneously. Kundrat has performed gastro-enterostomy for this condition, and H. Albrecht has proposed to open the abdomen and pull up the intestine from the pelvis and prevent its prolapse by means of suturing the appendices of the sigmoid flexure to the peritoneum of the bladder. E. H. L. O.

Systematic Tampon Drainage of the Small Pelvis to Prevent Post-operative Peritonitis.

E. SCHÜRMANN, Barmen (*Zentralb. f. Gyn.*, 1910, No. 30), recommends that, after every laparotomy, the whole of the peritoneal suture, even beyond the insertion of the ligamentum infundibulo-pelvicum on the rim of the pelvis, should be covered with iodoform gauze filling in the whole of the small pelvis. This tampon drainage has up to the present proved very satisfactory in thirty cases. Schürmann imagines the action of this drainage to be that of a mechanical stimulus to the reactive forces of the peritoneum, so that the infective germs, which need some time to develop their activity, find the peritoneum already in a defensive attitude. J. J. M.

Metastases of Cancer in the Ovaries and Pouch of Douglas.

ROSENSTIRN, Berlin (*Archiv f. kl. Chirurgie*, July 1910, Bd. 92, Heft 3), in investigating the mode of origin of metastases in the pouch of Douglas, has made minute microscopic examinations in 15 cases of cancer of the abdominal viscera. He concludes that metastases in the small pelvis may be considered as due to implantation. This view was especially supported by a case of carcinoma of the pancreas, in which, microscopically, small carcinomatous foci were found on the serosa of the pouch of Douglas, and no metastasis could be found elsewhere. He recommends that in radical operations for cancers of the peritoneal cavity, the lowest part of the wall of the pouch of Douglas should be removed. J. J. M.

The Physiological Importance of the Corpora Lutea of the Ovaries.

N. A. BIELOW (*Russky Wratsch*, 1910, No. 12), from experiments made in the laboratory of Professor A. Reprew, at the Institute for General and Experimental Pathology at the Charkow University, concludes—(1) that the corpus luteum represents a gland with temporary function of extraordinary importance in the economy of the female system; (2) the term "glandula lutea ovarii" should replace that of "corpus luteum"; (3) the secretion of the glandula lutea diminishes the blood pressure and the pulse rate; (4) in regard to its influence on blood pressure, the glandula lutea is antagonistic to the suprarenal capsule; (5) the pathology of the glandula lutea must be considered in regard to hypersecretion and hyposecretion, each of which must, in some way, react on the general system. J. J. M.

Pseudomucinous Cystoma of the Ovaries and Myxomatous Changes in the Peritoneum, the so-called Pseudomyxoma Peritonei, following the rupture of such cysts.

ERICH AHLSTROM (*Muenchener m. Wchns.*, 1910, No. 31, S. 1661), in a comprehensive monograph, discusses the above morbid processes, especially as regards their pathological anatomy, but also from a clinical point of view. In addition to the problems treated by previous authors, he draws attention to several points which have attracted less attention, such as (1) the ætiology of pseudomucinous cystoma; (2) the occurrence of myxomatous degeneration of the cyst wall; (3) the causes of rupture of pseudomucinous cysts; (4) the frequency of such cysts and the causes of their occurrence; and from the clinical point of view, he discusses their symptomatology, prognosis and, very fully, their surgical treatment. He concludes that pseudomucinous cysts, though anatomically of a typically innocent structure, must be considered as being on the borderland between innocent and malignant tumours. He considers the term pseudomyxoma peritonei less appropriate. J. J. M.

Acute Peritonitis apparently originating from the Female Genitalia.

G. PALLIN, Lund (*Zentralb. f. Gyn.*, 1910, No. 30), reports five cases of general peritonitis in women, the origin of which was doubtful. In four cases the appendix was out of the question, and probably so in the fifth. Pallin is inclined to attribute the infection to an external source, through the genitalia, though the latter showed no morbid alteration. This means an infection through the tube without any salpingitis; cases of the sort, though rare, have been met with. J. J. M.

Femoral Hernia of Fallopian Tube without Ovary.

CHARLES H. PARKES (*Journ. Amer. Med. Assoc.*, 1910, Vol. xlv, p. 649).—The patient was 33 years old, with a family and personal history which was devoid of interest. She complained of a small tumour in the region of Scarpa's triangle on the right side. The tumour had existed for about fifteen years, and varied in size and consistency from time to time, but caused no inconvenience. On examination the swelling was tense, and gave a sensation of slight fluctuation. No impulse was obtained on coughing, and no change in the size on pressure. An examination of the pelvis revealed nothing definite, because the parts appeared to be very tender. The sac was opened, and one-third of the Fallopian tube was discovered inside it. The tube was congested, purplish in colour, and could not be reduced without incising Gimbernat's ligament. The abdomen was subsequently opened, and the right ovary was found plastered up against the femoral ring, cystic and about the size of an orange. The right tube was removed and the parts were stitched up. C. N. L.

Clinical Reports of the Rotunda Hospital.

TWEEDY (*Dublin Journal of Med. Science*, July 1910), in this report, refers specially to eclampsia, contracted pelves and to the treatment of premature children. Up to date he has had 73 cases of eclampsia with 6 deaths, the last 26 without a death. The causation of the marked increase in the numbers, as many as those of his two predecessors combined, is not explained. Dr. Tweedy thinks that irritation of the digestive tract is an exciting cause of the convulsions. Among his cases he includes women without fits if they are unconscious and have albuminuria, while he excludes all women without albuminuria and cases of chronic renal disease. Eclamptics with embarrassed breathing are turned on the chest with the head hanging over the bed to free the air passages, and oxygen is administered along with artificial respiration where necessary. The stomach is not allowed to remain full.

Dr. Tweedy enters a plea for the estimation of the pelvic diameters in inches; thus, if 4¼ be taken as the normal true conjugate, we may institute five classes of deformity measuring the degrees by stages of half an inch. Precision in measurement can be carried out only by Skutsch's pelvimeter, the use of which requires considerable practice. He no longer considers it justifiable in a hospital to induce labour or to perform prophylactic version for uncomplicated contraction. Walcher's position can be maintained for many hours without inconvenience if the patient be permitted to rest her feet on a chair between the pains.

Premature children often shrivel up from lack of fluid after the first week. The lack of subcutaneous tissue renders hypodermic injection of saline impracticable. Rectal injections provide the only means whereby sufficient fluid may be absorbed; it is necessary to hold the child up by the heels. E. H. L. O.

Childbirth after apparent Menopause.

L. BUCKLE (*Journ. Amer. Med. Assoc.*, 1910, Vol. xlv, p. 568).—C.H., a well-preserved and healthy-looking woman, aged 50 years, always menstruated regularly, and never had any genital trouble until fifteen years ago. At that time she gave

birth to twins, making the number of children seven in all. About a year later menstruation returned, but with considerable irregularity, and in another year it ceased altogether. Two years ago, *i.e.*, eleven years after the cessation of menstruation, she began to feel out of sorts. She lost her appetite, had nausea and felt weak. Examined and treated by several physicians, her true conditon was not diagnosed. Subsequently definite signs of pregnancy appeared, and she was delivered of a normal child. C. N. L.

Changes in the Organs during Pregnancy in their Chinical Aspect

HOFBAUER, Königsberg (*Volkmann's Samml.*, No. 586, 1910) describes some of the changes in organs which occur normally in pregnancy. The first of these is a swelling and congestion of the mucosa of the larynx, especially of the interarytenoid space and of the false cords. This is seen early in pregnancy, especially in multiparæ, and is found in three-fourths of all pregnant women. It leads to hoarseness and false intonation in singers, as the cords may be partially or completely covered, and cannot be fully approximated from the swelling of the interarytenoid membrane. The epithelium is loosened and thrown into folds, and there is cell infiltration of the subepithelial tissue. Similar conditions have been found in the nose in pregnancy and also at menstrual periods. The importance of this condition is obvious, as it so closely simulates the appearance of early tuberculosis of the larynx, and Hofbauer seems to suggest that laryngologists have often made this mistake. He does not give much light on the subject of the alleged hypertrophy of the heart and increased blood pressure. The central veins of the liver are dilated and fatty infiltration follows, especially in the centre of the acini. After treatment with chloroform the fatty cells are seen vacuolated, while the nuclei remain and take stains well; the absence of glycogen from them is demonstrable also, as well as stagnation in the bile ducts. Chvostek maintains that the liver is palpably enlarged from congestion in the menstrual and pre-menstrual period. The power of assimilating sugar is diminished in 80 per cent. of women in the last third of pregnancy, also the power of converting amino acids into urea. The stagnation of the bile flow tends to cholelithiasis, though this is much more common during the puerperium than in pregnancy. According to v. Leyden there is a pregnancy kidney characterized by a change in the cells, while the interstitial tissue remains unaffected. There is no nephritis, the alteration being of a degenerative character, with cloudy swelling and fatty infiltration, especially in the convoluted tubules and Malpighian corpuscles, also according to some observers, but less markedly in the Henle loops. The nuclei remain, so that complete recovery is possible. The possible aggravation of the conditions of a pre-existing nephritis, whether interstitial, parenchymatous or glomerular, is accordingly evident, and their improvement after interruption of the pregnancy is explicable. The bladder itself is markedly hyperæmic with contortion of the veins, especially towards the neck. The tissues are infiltrated with cells chiefly lymphocytes, pseudoeosinophils and plasma cells, much as in the corresponding process in the larynx. Somewhat similar conditions with the corresponding frequency of micturition are found during the menstrual periods. Thus the liability of the lying-in woman to cystitis is explained. A like change occurs again during pregnancy and menstruation in the ureters, thus explaining the predisposition of women to pyelitis. Jolly has shown that out of 162 cases of dilatation of the ureter, 115 were preceded by puerperium or pregnancy, and the right ureter was the one affected in the ratio of 93·7. The conditions above described predispose to this, and the exciting cause is to be found in the displacement of the bladder causing a kink in the ureter. The aggregation of changes readily leads to the migration of bacteria, commonly the colon bacillus, and thus to a septic process. Thus the indications point to catheterization of the ureter as the most appropriate treatment. The complication

of an appendicitis during pregnancy has been frequently observed, in most cases probably a recrudescence of a previous condition. Waldeyer and Füth have shown that the uterus rising out of the pelvis in its growth pushes up the cæcum and appendix, so that should a purulent collection form in the region of the appendix it is more likely to burst into the peritoneal cavity than to burrow downwards towards the pelvic outlet. Whether pregnancy produces a hyperæmic condition of the appendicular mucosa is still doubtful, but several observations incline one to think it possible. The loosening and desquamation of the superficial layers of the vaginal epithelium in pregnancy open the way to gonococcal infection. The alterations of the glands with internal secretion are important, but too little is known of their functions to enable dogmatic assertions to be made. The thyroid is palpably enlarged, and recent observations show the pituitary body also to be enlarged in its glandular anterior portion. The cells become permanently affected, so that previous pregnancy can be diagnosed from them. So also the suprarenal gland cortex becomes hyperplastic, as it does in the compensatory hypertrophy after castration, while in eclampsia the chromophile elements are reduced. Adrenalin production is increased, as may be seen by the marked aggravation of the symptoms during pregnancy in exophthalmic goitre. The changes in the skeleton may be ascribed to the action of the internal secretions as well as the changes in carbohydrate metabolism. Removal of part of the parathyroid leads to tetany in pregnant animals. Hofbauer concludes with a description of the changes in the blood; neutrophil polyneucleosis disappearing after delivery, increase in the blood content which must be established to prevent abortion, increase in the total quantity of the blood, increase in the fibrin percentage and of chlorides are mentioned. Increase in the antitrypsin content of the serum, beginning in the first months of pregnancy, and especially marked in eclampsia and lipæmia, are also mentioned. A copious literature is appended. E. H. L. O.

Changes in the Upper Air Passages during Pregnancy, Labour, and Childbed.

Imhofer, Prague (*Muenchener m. Wchns.*, 1910, No. 31, S. 1665), in a communication to the Dresden meeting of German laryngologists, drew the following conclusions:—During pregnancy and childbed the upper air passages, the nose and larynx, especially the posterior wall of the latter, are undoubtedly apt to become swollen; these tumefactions gradually disappear during childbed, and in this the duration of the supine position seems to have some influence. During labour itself the upper air passages as a rule undergo no change; it is only in prolonged and difficult labours that hyperæmia and congestion of the larynx occurs. There is no reason whatever to suppose the swelling of the posterior wall of the larynx, in spite of its resemblance to tuberculous infiltration, to be due to tuberculosis or to be a predisposing factor therefor. Hæmorrhages in the upper air passages do, in some cases, occur during pregnancy or during or shortly after labour, but by no means so frequently as is suggested by the literature on the subject. Paresis of the laryngeal muscles cannot be taken as directly due to pregnancy except in exceptional cases. The practical importance of these conclusions is that, in cases in which some narrowing of the larynx already exists (in which tuberculous infiltration is the first thing to be considered), an alarming increase in such narrowing may occur during pregnancy, yet in practice this danger is not to be looked upon too seriously; from a practical point of view the possibility of a wrong diagnosis, especially of one of tuberculosis, is of far more importance. The ordinary changes in the upper air passages, in pregnancy, labour and childbed, evidently depend on purely mechanical conditions. J. J. M.

Pregnancy and Laryngeal Tuberculosis.

A. von Sokolowsky, Warsaw (*Zeitschr. f. Laryngol., Rhinol. u. ihre Grenzgebiete*, Bd. ii, Heft 6), finds, contrary to general opinion, cases of laryngeal tuberculosis sometimes, though rarely, pass through pregnancy without serious consequences for mother or child. It is, however, extremely important to recognize commencing or latent tuberculosis of the larynx early in pregnancy, and cough or difficulty in swallowing are not to be passed over as merely troubles of pregnancy, but demand laryngoscopic examination. At the same time, one must take precautions against a wrong diagnosis of the disease, by careful objective examination, the use of Calmette's reaction, etc., for many women desire abortion and strive to deceive their physician. The treatment is not a simple affair; each case must be treated individually; in commencing tuberculosis, when the pregnancy is well borne without any fever in the early months, the pregnancy should not be terminated, but the patient should be placed under good hygienic conditions and kept under observation. In poor people when, from the expense, this is not possible, abortion should be induced, for the disease must be aggravated by the pregnancy and confinement under unfavourable conditions. Even in commencing tuberculosis, if there is persistent fever, depraved general condition, and the pregnancy is being badly borne, interference is necessary, especially if there be hereditary tuberculous taint; this is also the case in the so-called fibrous form of pulmonary tuberculosis, in which, as a rule, pregnancy causes great distress and leads to dyspnœa and progressive weakness of the heart. J. J. M.

Myomata in Pregnancy, Labour and Childbed, and their influence on Conception.

F. von Winckel (*Muenchener m. Wchns.*, 1910, No. 32, S. 1698) writes a warm appreciation of a monograph by Theodor Landau on "myomata in pregnancy, labour and childbed," a subject discussed at the Congress at Buda Pest last year after masterly reports by Schauta and Engström (*ante* Vol. xvi, p. 414). In 1901, in a work on "The Treatment of pregnancy and labour complicated by myomata," Landau summarized all that was then known on the subject and formulated principles as to its pathology and treatment which, broadly speaking, are still generally accepted. The ample material the brothers Landau have had at their command, more ample perhaps than any other clinic, has given them exceptional opportunity for the clinical and anatomical study of this complication of myomatosis uteri, and for the successful development of the technique of its surgical treatment. This development has been gradual, for, while in 1885, Gusserow found that of 395 operations for myoma 122 proved fatal, in 1897 Olshausen 45 fatal in 806, a mortality of 5·6, and Winter's statistics of the abdominal supravaginal amputation, in 1904, showed 32 deaths in 389 cases, a mortality of 4·6, in 283 operations by the brothers Landau in the last five years there have been only two fatal operations, No. 90 and No. 131, of the series.

v. Winckel gives Landau's conclusions on the relation of myomatosis uteri to fertility pretty fully, and they are most interesting. Contrary to the ideas formerly held, that the subjective and objective symptoms of myoma usually appear after a woman is 30 or 40 years old, Landau has found myomatosis uteri to be essentially a disease of youthful sexual life; in 400 cases of large fibrous tumours operated upon for severe symptoms, 42 were found in women between 20 and 30 years old.

As long as the way is free for the sperma and ovum to meet, and a minimum of endometrium is available for the nidation of the ovum, the number and size of the tumours, and even their anatomical condition do not interfere with the occurrence of pregnancy, and Landau was not surprised, on examining his cases of myoma in regard to fertility, to find that most of them had been fertile; of the last 250 cases, 182 had been, and 68 had not been, pregnant. Of the 68, a few only (15) had even

slight changes in the perimetrium, to which indeed as a cause of sterility little importance need be attached. It is, however, shown by these statistics that among the 68 sterile cases there were only 40 in which the myoma was the only pathological condition; in the other 28 some other factor must be considered as a possible cause of the sterility, and the mere fact that of the women upon whom operation was necessitated by myoma, three-fourths had been pregnant, shows that the alleged tendency of the presence of myoma to cause sterility can only exist in a small proportion of the cases. Moreover, it appears from the anamneses that the fertile myomatous women who underwent operation exhibited adnexal changes similar and to the same extent as those found in the unfertile, so that the presence of myomata in the womb has no effect whatever upon conception. It is therefore unnecessary to prove that pregnancy has been found by various observers associated with myomata in every possible situation in the uterus, of every possible size, with solitary and with multiple tumours.

It may be asked whether such tumours may not occasionally diminish the capability of conception in a proportion of the cases, that is, whether pregnancy only occurs in myomatous patients in spite of the tumour. Landau considers this question too wide to be considered. All myomata are intramural at first, and it is by the contraction of the muscular walls of the uterus that some become submucous, while even intramural tumours do not remain in one place, but migrate in their softer surroundings, and it can be easily understood that myomata of a certain development may come to obstruct the uterine canal, and, after a further migration, leave it free.

Even such an exact statement of the percentage of sterility with myoma does not settle the matter, for daily observation constantly shows us myomatous women who, according to theoretical views and suppositions, should be incapable of fertilization, and are nevertheless pregnant; so that for the individual case nothing can be concluded from a general principle, and no prognosis is possible. On these grounds also we must admit that myomatous women may marry. J. J. M.

Excess of Chlorides in the Blood in Pregnancy and Eclampsia.

Javal (*La Gynécologie*, March 1910), who has worked with Widal at the chlorides in the blood of those suffering from Bright's disease, has extended his investigations to ten cases of eclampsia in the clinique of M. Doleris at the Saint-Antoine Maternity. He bled the patients and analyzed the chlorides in the serum that separated from the naturally formed clot. The observations were controlled by tests of the blood of normal women at term as well as of many patients in his previous work. He employed Volhard's method of estimating the chlorides in the ash of the serum, and Kjeldhal's method of estimating the nitrogen, Yvon's for urea after precipitation of the urea, and the albumen was weighed. The ten cases are given in detail. All, except one, had, during the crisis of the attacks, a manifest hypertonicity of the blood. The albumen content was not affected, and the albuminuria does not appear to diminish the albumen in the blood serum. The nitrogen, as evidenced by the quantity of urea, was slightly increased. The chlorides were markedly increased, but they are so in normal pregnancy, as has already been stated by Bar, and they were found to diminish rapidly after the attacks. The excess of chlorides may by osmosis tend to increase the pulse tension, and also render the blood more liable to take up toxic substances. Without attacking any of the hypotheses formulated on the production of eclampsia there is no doubt, says Javal, that there is some connection between the excess of chlorides and eclampsia. E. H. L. O.

An Acute Anæmia (? Pernicious Anæmia) Complicating Pregnancy.

Audebert and Dalous (*Ann. de Gyn. et d'Obstét.*, August 1910) record a case of pregnancy complicated by a severe anæmia, similar in many respects to pernicious

anæmia. The patient was 26 years of age, and had had two full-term children without special trouble. During the early part of the third pregnancy anorexia and vomiting were marked, and during the last month the patient was incapacitated by extreme muscular weakness and anæmia. About the same time the legs and eyelids became œdematous. On examination, the usual signs of extreme anæmia were obvious. The spleen was enlarged to twice the size of the folded fist. The urine report was: quantity, 1,600 grammes; acid; s.g. 1012; urea, 11·01 grammes per litre; albumen, 0·25 grammes per litre; sugar, *nil*. The blood examination showed red corpuscles, 643,150; leucocytes, 12,632; hæmatoblasts, 64,000. The leucocytes consisted of:—

Polynuclear—neutrophiles	63·0
Polynuclear—eosinophiles	3·3
Lymphocytes	21·3
Mononuclear cells	6·2
Nucleated red cells	6·2

The patient was given cacodylate of soda daily, and a fortnight later, although severe epistaxis had repeatedly taken place, the œdema was less, and the urine only contained a faint trace of albumen. Eighteen days after admission to the hospital a macerated child was born. The blood lost during delivery was less than usual. Delivery was immediately followed by improvement in the patient's symptoms and appearance, but myelocytes (2·7 per cent.) were repeatedly found in the blood. Five days after delivery the red cells numbered 1,448,000, the leucocytes 8,531 and the hæmatoblasts 440,000. Ten days after delivery the numbers were respectively 1,653,460, 5,759 and 488,000. Six weeks after delivery the red cells had gone up to 2,066,460 and the leucocytes had dropped to 5,470.

The authors admit that some of the symptoms may have been of renal origin, but think the blood counts and splenic enlargement exclude the renal lesion being the only one; they regard the case as being of a pernicious anæmic type, and lay stress on the immediate improvement brought about by evacuation of the uterus. In two patients with pernicious anæmia Tarnier induced labour at the seventh and fourth month—both died. Garipuy and Claude also record a fatal case, but Chiara and Clivio have had good results from early induction of labour. C. W.

Hermeralopia in Pregnant and Nursing Women.

Garzia del Mazo (*Rev. de Med. y. Cir. Pract.*, May 14 1910) reports the following cases:—(1) A woman, 28 years of age, had had two normal pregnancies, after each of which she suffered from hemeralopia while nursing, but recovered when the child was weaned. In daylight her acuteness of vision, field of view and sense of colour were quite normal, as was also the ophthalmoscopic condition. (2) The other patient, 25 years old, had had four normal pregnancies, in each of which she suffered from the seventh month, from hemeralopia, which disappeared during the childbed; on the last occasion, however, it recurred after six weeks' nursing, and again disappeared when the child was weaned. J. J. M.

Incarceration of the Pregnant Uterus.

R. W. Lobenstine (*Bulletin of the Lying-in Hospital of New York*, Vol. vi, No. 2) publishes an interesting paper on this subject.

Ætiology. Pregnancy may start in a uterus retrodisplaced by old adhesions, fibromyomata, relaxed pelvic floor, or the uterus may become displaced during the earlier months of pregnancy by hard work, heavy lifting or possibly by carelessness of the patient in allowing the bladder to be persistently overfilled. In the majority of cases the uterus will right itself, but not in all. The percentage of cases in which this happens can only be a matter of conjecture.

Mechanism of spontaneous reposition. The most important factor is the "intermittent contracting power" of the uterus. By this means the overstretched anterior wall draws itself together, so to speak, and pulls the fundus upwards and forwards. Next in importance is sacculation whereby the anterior wall bulges upwards and gradually draws the fundus after it. This mechanism is especially noticeable in those cases where the ovum is embedded in the anterior uterine wall. The action of the round ligaments is disputed, but the author thinks they must help to produce and maintain anteversion of the growing uterus. One clinical point is noted: if the cervix rides high above the symphysis, spontaneous replacement is unlikely to recur.

Symptoms are always referable to the bladder. The author describes illustrative cases of the four urinary conditions which may arise, viz., (*a*) retention of urine with idiopathic hæmorrhage from the bladder; (*b*) retention of urine with hæmaturia after catheterization; (*c*) rupture of the bladder; (*d*) gangrene of the bladder.

Treatment. Before incarceration it is usually sufficient to warn the patient of the dangers of allowing the bladder to become overfilled. It is advisable for the patient to wear a pessary for the first four months. When symptoms of incarceration are present the bladder must be emptied and kept empty. Under anæsthesia, attempts at reposition should be made. If successful a large ring pessary should be inserted. If unsuccessful two courses are open, either posterior colpotomy on the chance that the fundus is held down by adhesions, or laparotomy by which means the uterus can be brought out of the pelvis. A third method which should never be employed except in cases of "active abortion," is emptying the uterus. The author thinks the safest method would be to incise the uterus through the posterior vaginal fornix. J. A. W.

Axial Torsion of the Pregnant Uterus.

Glinski, Cracow (*Monats. f. Geb. u. Gyn.*, Bd. xxxi, Heft 4), reports the sudden death of a multipara, aged 33, in the ninth month of pregnancy owing to axial torsion of the uterus. The seat of the torsion was at the cervix. There was a quantity of clotted blood beneath the detached placenta. In the absence of any complication from tumours or otherwise, the cause of the torsion must have lain in the length of the cervix and the laxity of the corpus; the normal torsion of the uterus and the bulging of the uterine wall due to the attachment of the placenta, also severe exertion shortly before the accident, must also be taken into account. J. J. M.

Fixed Ovarian Tumours which prevent the Rising of the Pregnant Uterus.

Saltzman, Jun. (*Engström's Mitteilungen*, viii, 2, 1910) relates two cases in which torsion of the pedicle had produced adhesions which fixed the tumour and prevented the pregnant uterus from rising out of the pelvis. In one case the twisting of the pedicle occurred during pregnancy, in the other before conception. Both patients were relieved by operation, and the uterus in each was found quite unadherent. Both patients went to term, and were delivered successfully. E. H. L. O.

Rupture of the Uterus during Labour.

R. W. Lobenstine (*Bulletin of the Lying in Hospital of New York*, Vol. vi, No. 2) reports on forty-six cases of complete, twenty-nine of incomplete, rupture of the uterus, as well as three cases of rupture of the vaginal vault without actual laceration of the uterus, which occurred in a series of 60,000 labours. These figures confirm the observations of others, that a complete rupture of the uterus is a far more common injury than an incomplete, a possible explanation being that the incomplete are not always diagnosed.

Ætiology. Complete rupture of the uterus is seldom seen among the wealthy,

and is far more common in multiparæ than in primiparæ in the proportion of forty-four to five (the latter including three cases of rupture of the vaginal vault), whereas in the incomplete type the ratio was eleven to eighteen. The frequency of complete rupture in multiparæ is due to damage to the musculature of the uterus by past inflammation and cicatricial tissue, as well as to the fact that the general nutrition of a multiparous woman is not so good as that of a woman pregnant for the first time.

The following is an interesting table of the cause of rupture in the forty-six cases of the complete type :—

1. Spontaneous rupture due to	
Pelvic contraction	21
Scar of previous Cæsarean section	2
Following amputation of cervix	1
Transverse presentations	3
Hydrocephalus	1
2. Traumatic ruptures—	
High forceps	3
Internal version	3
Accouchement forcé	
for placenta prævia	5
for eclapmsia	1
Embryotomy	1
3. Ruptures due to combination of transverse presentations and internal version	3
Total	46

Pathological Anatomy.

There are two types of complete rupture of the uterus—the transverse and the longitudinal; the former usually is in the anterior wall, and the bladder is frequently extensively damaged. It is the common type of spontaneous rupture. The longitudinal, on the other hand, is usually lateral and involves the uterine vessels, and is the type of traumatic rupture. In the series of cases under review, twenty were probably transverse in origin, and twenty-six longitudinal. The majority of cases showed more or less combination of the two types, probably the result of excessive trauma.

Mechanism of spontaneous rupture. Spontaneous rupture of the uterus, with very few exceptions, is the result of tonic contraction. The site of rupture depends on whether the presenting part has passed the lower uterine segment or not. In those cases where the head imprisons the cervix against the pelvic wall the rupture will take place through uterine tissue. If the lower uterine segment is drawn up over the presenting part (*e.g.*, shoulder presentation) the rupture will take place through the vaginal vault and may extend into the lower uterine segment secondarily. Probably the hypertrophied broad ligaments act as definite supports to a dangerously thinned lower uterine segment.

Symptoms and diagnosis of complete rupture of the uterus are straightforward in the majority of cases. Sudden pain, equally sudden cessation of the hitherto violent uterine contractions with the symptoms of shock, which are usually rapid but may be insidious in their onset, can only mean uterine rupture. Associated with these symptoms is the retraction of the presenting part. If the fœtus has entirely escaped it will be plainly felt lying beside the uterus, which is large and flabby and not firmly contracted as is so often stated. There may, or may not, be visible hæmorrhage. Death may rapidly ensue from loss of blood; if the patient survives the first twenty-four hours a rapidly progressing and virulent sepsis may prove fatal.

Incomplete rupture of the uterus is a less common and less fatal variety.

The common type is a longitudinal tear into the broad ligament starting from the vaginal vault or cervix. The next most common type is a transverse one thorugh the cervix into the utero-vesical pouch. Incomplete ruptures higher up in the uterus are uncommon and much less serious. Incomplete rupture of the uterus is more frequently due to traumatism, especially the rapid dilatation of the cervix. But of twenty-nine cases, twenty-four were due to rapid dilatation and extraction for placenta prævia, eclampsia or accidental hæmorrhage. The worst lacerations followed the use of the Bossi dilator. The most frequent cause of spontaneous rupture is the yielding of old scar tissue in the cervix or vaginal vault.

Mortality. In the forty-six cases of complete rupture there were thirty-four deaths, 73 per cent. In three cases of rupture of the vaginal vault there was one death, 33 per cent. Of the twenty-nine cases of incomplete rupture, eight died, 25 per cent. Under this last heading the deaths occurred in cases of eclampsia and central placenta prævia, both of which independently have a fairly high mortality.

Treatment. That prevention is better than cure is particularly true of rupture of the uterus, and the author emphasizes this point in nine maxims. The treatment of the actual rupture depends both on its extent and on the surroundings of the patient. Incomplete ruptures and those of the vaginal vault should in all cases be treated by plugging with or without suture. The only indication for abdominal section is hæmorrhage persisting or recurring after plugging. The ideal treatment of complete rupture is abdominal section with suture of the rent or hysterectomy. But this needs ideal surroundings. If the diagnosis is made early with the child still in the uterus it is far better to transfer the patient to hospital undelivered. Delivery can then be effected either *per vias naturales* or by an abdominal section, with every means at hand of applying immediate and appropriate treatment to the actual rupture. But as all attempts at delivery *per vaginam*, whether successful or not, will certainly increase the size of the rent and the amount of shock, the difficulties of the cases will be much increased if delivery is effected in the patient's own home. If the child and placenta have escaped into the peritoneum immediate abdominal section is the only method of treatment, but the outlook is very bad.

On the whole the author inclines to abdominal section as the correct treatment except (*a*) in clean cases with small ruptures where the hæmorrhage can be controlled by plugging; in such, operation is unnecessary, and (*b*) bad cases with severe shock, where no operation can be of any value. No diminution in the mortality can be expected until a diagnosis is early and confidently made and treatment started as soon as the diagnosis is made. J. A. W.

The Treatment of Rupture of the Uterus in Connection with Seven Personal Observations.

Issel, Freiburg (*Monats. f. Geb. u. Gyn.*, Bd. xxxi, Heft 4), states that at the Freiburg Klinik rupture of the uterus is treated surgically, the operation preferred being removal of the organ by laparotomy. Six of the reported cases were operated on: four by total extirpation, one by supravaginal amputation, one by suture of the laceration. When the rupture is high up and the patient drained of blood, amputation, as a shorter proceeding, should be preferred, otherwise total extirpation. Suture of the rupture increases the danger of infection, and the cicatrix is a danger in subsequent pregnancies. In private practice the patient should be sent into a clinic without any attempt to deliver her; if this is not possible she should be cautiously delivered, and the wound in her uterus drained. J. J. M.

Vaginal Cæsarean Section as a Method of Artificial Abortion or Premature Labour.

Klein (*Muenchener m. Wchns.*, 1910, No. 31, So. 1667) reported to the Munich Gynæcological Society, that he had induced abortion in 35 cases, but never without

consultation; 18 times for tuberculosis; twice for hyperemesis; 11 times for nephritis and pyelitis, and in the rest for osteomalacia or some other indication. In his earlier cases he had begun by introducing a laminaria tent, but in one-fourth this caused high fever. He had also had experience with the metreurynter and with tamponade, and now recommended in all cases in which artificial abortion was indicated, anterior hysterotomy as the method of choice, on account of its rapidity and security from infection. He has operated in this way in 7 cases without any mischance. J. J. M.

JAHREISS, Augsburg (*Zentralb. f. Gyn.*, 1910, No. 32), recommends anterior vaginal hysterotomy as the simplest and least harmful method of emptying the uterus in the earlier months of pregnancy. According to his experience atony of the uterus is not to be dreaded. After emptying the uterus he plugs the cavity with iodoform gauze, sutures the cervix, and replaces and fixes the displaced bladder, then plugs the vagina, gives an injection of ergotin, and applies pressure with a bag of sand. He gives details of four illustrative cases. J. J. M.

Præfœtal Dilatation of the Vagina in Breech Presentation.

SAUVAGE (*Soc. d'Obst. de Gyn. et de Pæd.*, 1910, Vol. xii, p. 195).—The author bases his paper upon a series of 50 cases, and his views may be summarized as follow :—

1. Præ-fœtal vaginal dilatation is not always certain in its action, but it usually results in the acceleration of labour. It has a constant influence upon the duration of the second stage of labour, which, provided the expulsive forces are normal, is very much shortened. When interference is necessary it is made easier by the dilated condition of the soft parts.

2. The infantile mortality is in consequence much reduced, both during labour and the days following; the vitality of the infant is greater, for it has not undergone compression by the soft parts. This is more especially so in primiparæ.

3. The method is advisable in all cases of breech presentation occurring in primigravidæ when the child is viable. It can be extended with advantage to those cases in multiparæ in which the baby presents by the breech and is very large.

4. The best results are obtained by placing a Champetier de Ribes' bag in the vagina, and by dilating this to the size of the palm of the hand. The bag is left to be spontaneously expelled. J. B.

Hebosteotomy in Patient's Home.

SCHWAB (*Muenchener m. Wchns.*, 1910, No. 34, S. 1813) reported the following case to the Nuremberg Medical Society :—A ricketty secundipara, aged 30, the wife of a small shopkeeper, with a contracted pelvis of the first degree, whose previous confinement in the University Fruenklinik had, after vain attempts at version, been terminated by perforation of the child, had had strong pains for 30 hours without the engagement of the head; the membranes had ruptured 24 hours before Schwab performed hebosteotomy by Döderlein's method, at the house of the patient. The condition of the child led to its extraction by forceps an hour and a half later. The child was alive and did well; the mother was not injured; bladder and vagina intact and no hæmatoma. Childbed untroubled. She was able to move about without any difficulty from the eleventh day. J. J. M.

The Diminution in the Pulse Rate in Childbed and its Cause.

LEWISOHN, Berlin (*Monats. f. Geb. u. Gyn.*, Bd. xxxi, Heft 4) has found, from experiment, that in pregnancy and childbed the pulse-rate and temperature are independent. Slowing of the pulse is a symptom proper to childbed, and, owing to the great instability of the pulse in childbed, bradycardia is often unnoticed.

Severe labours, premature and false births, have a retarding influence on the bradycardia, which is a purely physiological symptom, only to be expected in the childbed of healthy patients. When they get up it disappears, often temporarily only, or it may then only definitely appear. The blood pressure then generally sinks also. Lewisohn supposes the cause of the bradycardia to be the diminished flow of blood to the heart after delivery whereby the automatic stimulus of the cardiac muscle to contraction does not recur so rapidly. J. J. M.

Formation of Multiple Abscesses in a Puerperal Uterus.

Beyer, Halle (*Monats. f. Geb. u. Gyn.*, Bd. xxxi, Heft 4) reports the formation of perimetric abscesses originating from the uterus of a primipara, aged 33, four and a half weeks after delivery. She recovered after evacuation of the abscess and extirpation of the uterus, in the wall of which were many purulent foci. The pus contained non-virulent hæmolytic streptococci. J. J. M.

Successful Removal of Large Sacro-coccygeal Dermoid on the Sixth Day after Delivery.

Mouchet (*Ann. de Gyn. et d'Obstét.*, August 1910) gives details of a remarkable case. He removed a cystic growth, weighing 1,800 grammes, from a chlid whose total weight was only 3,800 grammes before the operation. The child did well, and suffered but little from the shock of the operation. The cyst was attached in the sacral region, but did not actually involve the sacrum or communicate with the spinal meninges. It was 34 centimetres in circumference at its largest part. There were four cyst cavities, three of which contained clear yellow fluid, while the fourth looked like "erectile tissue" with turbid fluid, and contained 6 teeth and a few hairs. No histological examination was made. C. W.

The Time of Rising after Confinement.

Mme. Darcanne-Mouroux (*Soc. d'Obst. de Gyn. et de Pæd. de Paris*, 1910, Vol. xii., p. 188).—The study of the question of early rising after labour has resulted in Germany in the publication of statistics, which are in favour of this question. M.M. Alvensleben of Keil and Meyer of Marburg, each from a consideration of 100 cases, state that it is better for a woman to get up from the 1st to the 4th day after a normal labour. In Brittany, in the department of Ille and Vilaive, the peasant class regard it as a duty and a point of honour to rise from the 2nd to the 8th day after normal labour. The author has been able, during two consecutive years, to make an examination of these women, and she has obtained the following results. The pelvic organs were normal in 27 cases; subinvolution, 22 cases; prolapse second degree, 20 cases; retroversion, 13 cases; acute anteflexion, 6 cases; cystocele, 6 cases; pelvic inflammation, 5 cases; thrombosis, 1 case. Of these 100 cases who got up after labour from the 2nd to the 8th day 73 presented some pelvic lesion; therefore it seems that there is no indication whatever to modify our views as to the date of rising, or to advise rising during the first week after confinement. J. B.

Early Rising after Delivery.

D. v. Velitz, Pressburg (*Zentralb. f. Gyn.*, 1910, No. 25) has tested the practice of getting patients up soon after delivery as recommended by Küstner and Bumm on 496 puerperæ in his wards, and is very content with the results. Sound puerperæ were allowed to move and sit up in bed from as early as the third day—but not to stand up before the fifth. He found that under this practice the uterus diminished very rapidly in size, and the lochial discharge soon stopped. There was no instance of thrombosis or embolism. J. J. M.

Fibrous Tumour of the Kidney in a Fœtus accompanied by Hydramnios.

Funck-Bretano and G. Durante (*Soc. d'Obstet de Gyn. et de Pæd.*, 1910, Vol. xii, p. 174).—The patient was aged 30 years, and was 7½ months advanced in her ninth pregnancy. Owing to pressure symptoms the pregnancy was artificially terminated by rupture of the membranes. The quantity of liquor amnii which escaped was 11 litres. There was no post partum hæmorrhage. The infant died shortly after birth, and on post mortem examination the left kidney was found enlarged to the size of a small orange by a fibrous tumour, situated at the upper pole and distinct from the suprarenal capsule. J. B.

Normal Human Blood Serum as a Curative Agent in Hæmophilia Neonatorum.

J. E. Welch (*Amer. Journ. of Med. Sciences*, Vol. cxxxix, No. 6) describes a new form of treatment of this very fatal infantile disease. Treated by the ordinary medical measures, no less than seventeen out of eighteen consecutive cases died. Under the serum treatment twelve consecutive cases recovered. All the cases occurred in the New York Lying-in Hospital.

The ætiology of the disease is unknown. It commonly commences during the first week of life, usually on the third or fourth day. The primary bleeding may be from the skin or any mucous membrane. Post mortem, extensive hæmorrhages are found in the brain and liver. Usually there is no evidence of traumatism. Often the first bleeding is a slight oozing from the cord at its junction with the skin (*i.e.*, not due to faulty ligaturing) which persists despite all local remedies until a considerable quantity of blood has been lost. Other sources are the gastro-intestinal tract (hæmatemesis, melæna) or the lips and gums. Subcutaneous hæmatomata often of large size are seen which have no relation whatever to injuries.

Various theories have been expounded to account for this condition. Probably three factors either combined or separate will explain most instances of it. Some cases are a definite septicæmia. In others, especially when the hæmorrhage comes from the gastro-intestinal tract, the alimentary canal is blocked with decaying blood which must render digestion and absorption of food practically impossible—i.e., starvation. In others again there is probably a deficient coagulability of the blood.

The injection of normal human serum would counteract all these factors. It is certainly bactericidal as is shown by its great value in puerperal septicæmia. It is a perfect form of food ready to be absorbed and utilized. Lastly it is probable that a thrombokinase is supplied as is suggested by Kottmann and Lidsley. Further it is never toxic, thus differing from animal sera.

As regards dosage this must depend on the urgency of the case. It is advisable to begin with at least 10 cc. and repeat three times a day if the infant is bleeding only moderately. In severe cases it should be administered every two hours and in larger quantities if thought advisable.

The blood is easily collected by means of a very simple apparatus which is fully described in the paper.

In conclusion a full account of nine cases treated by this method is given.

J. A. W.

Injuries of the Testicles During Birth.

Simmonds (*Muenchener m. Wchns.*, 1910, No. 25, S. 1367), in a communication to the Hamburg Medical Society, pointed out that though little has been written on the subject, hæmorrhages into the tissue of the testicles are very common in children born dead or even alive. Though more frequent in breech cases they occur also in those born head first. The amount of hæmorrhage is very variable; some only to be

detected with the microscope, some visible to the naked eye, as multiple spots of blood, and some leading to tense infarction of the entire organ. In the last case the testicle is enlarged, tightly distended and of a dark reddish-blue colour, and the epididymis is also infiltrated with blood. Both testicles are affected in most cases, sometimes only one.

If the children die during or soon after birth the microscope merely shows separation of otherwise normal canals by the extravasation. In children who live longer, groups of canals are sometimes found in the extravasation, showing signs of commencing necrosis of their epithelium. Occasionally in infants of more advanced age one finds residua of the bleeding in large accumulations of chalybeate pigment in the interstitial tissue. Some caution is required in the explanation of these heaps of chalybeate pigment, for in atrophic and cachetic infants siderosis even in the testicles is not uncommon. Gross permanent changes in the organ due to injury during birth Simmonds has only detected once: in a boy of 4 who died from scarlet fever there was a circumscribed fibrous focus in the middle of his testicular tissue which could not be explained in any other way.

The cause of testicular hæmorrhages in the newborn is to be found in the compression of the testicle and spermatic cord during birth, but in nearly all cases of the kind the asphyxia commonly present is a contributing factor, and in cases born head first, testicular hæmorrhages may be accepted as a symptom of asphyxia.

PLATE, in the discussion on the above, suggested that in cases born head first, the vulval ring, tightly encircling the body after the birth of the head, as descent went on forced the blood towards the pelvis of the child. J. J. M.

Gangrenous Lymphangitis of the Scrotum in the Newborn.

LILLA (*Muenchener m. Wchns.*, 1910, No. 27, S. 1468) says that though this affection is very rare, it has attracted a good deal of clinical study, and is also identified on ætiologic and pathogenetic grounds. In a recent work Andrei discussed the 11 cases hitherto recorded in literature, and came to the conclusion that the port of entry of the specific agent, which he takes to be the streptococcus, is always some slight lesion of continuity in the healing of the divided umbilical cord.

Lilla, however, reports a case, in a child 28 days old, originating from a furunculous inflammation of the lower part of the abdomen. As a matter of fact, though the view of Andrei above stated may be correct for most cases of scrotal gangrene in the new-born, there does not seem to be any reason why, in quite young children under unfavourable conditions as to nourishment and hygiene, the infecting agent may not find some other port of entry. J. J. M.

A Case of Thoracopagus.

H. JENTTER, St. Petersburg (*Zentralb. f. Gyn.*, 1910, No. 25), delivered an octipara, aged 37, of a thoracopagus after division of the tissue uniting the twins, which together weighed 4,250 gms. The umbilical cord was single with a velamentous insertion, and contained three vessels, one vein and two arteries. The placenta was single. The monster was a sternopagus, or thoracopagus dicephalus tetrabrachius tetrapus. In a common pericardium there were two hearts; there were four lungs, but only one liver. The author discusses the mechanism of labour in such cases.

J. J. M.

Obliteration of the Bile Ducts in an Infant.

MERLE and PETIT (*Ann. de Gyn. et d'Obstét.*, August 1910) record the results of an autopsy on an infant 9 weeks old. The history of the case was that at birth (at full term) the child weighed 2,450 grammes and appeared to be normal. Mother healthy—no syphilitic history. At the end of the second week jaundice came on

and gradually increased. From birth the motions had been quite colourless, but the urine was deeply pigmented. On palpation the liver edge could just be felt; the spleen was not enlarged. The child died at the end of the 9th week, having had a severe hæmorrhage from the rectum two days before death. The temperature was normal throughout.

At the autopsy there was nothing remarkable except the state of the hepatic system, where the following condition was found:—The liver weighed 170 grammes, and was dark-green in colour; the duct of Wirsung and the ampulla of Vater were normal, but the duct of Santorini could not be found. The common bile duct was represented by a thin fibrous thread leading towards the pancreas; in the small omentum it could not be distinguished, although the hepatic and portal vessels were present in the usual postion. The gall-bladder was only 10 millimetres long, and ended posteriorly in a fibrous cord which followed the portal vein, but ended by joining the obliterated umbilical vein.

On microscopic examination the vessels showed signs of inflammation, but were otherwise normal. All that could be found to represent bile-ducts were small flattened spaces lined by degenerate epithelium. The whole was surrounded by dense fibrous tissue and inflammatory round-celled infiltration. The liver cells were comparatively healthy, but the connective-tissue was universally inflamed.

The authors regard the case as one of fœtal angiocholitis, which destroyed the bile-ducts but left the liver-cells sufficiently healthy to support life nine weeks.

C. W.

Fibrous Tumours of the Kidney in a Fœtus.

Funck-Brentano and Durante (*La Gynécologie*, March 1910) describe a fibrous tumour of the left kidney of a fœtus which died a few hours after birth. The histological characters are described in detail, with the aid of micro-photographs, and they are such as to lead the reporters to believe that the growth of fibrous tissue started in the cicatrix of an intrauterine injury rather than in a new growth in the ordinary acceptation of the term. The woman suffered from hydramnios to such an extent as to necessitate the induction of labour. Liquor amnii was *collected* to the extent of eleven litres.

E. H. L. O.

Thèses.

Uterine Inversion. Beyly. May 1910. Bordeaux.

L'embryotomie rachidienne en écharpe. Joby. May 1910. Lyon.

Sialorrhœa. Besnard. May 1910. Paris.

Adéno-phlegmons iliaques. Bonvoisin. June 1910. Paris.

Vaginal hysterectomy (Schuchard-Schauta operation). Ehrenpreis. May 1910. Paris.

De l'avenir des femmes ayant eu des pleurésies avant ou après la puberté et devenant mères. Mme. Libin. May 1910. Paris.

Evolution du moignon [stump] cervical après l'hysterectomie abdominale subtotale. Aymeric. May 1910. Toulouse.

Anémies pernicieuses de la grossesse (a case of marked improvement after delivery). Magnes. June 1910. Toulouse.

E. H. L. O.